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THE Journal OF Nervous and Mental Disease

Original Articles

ON SPASTIC PARALYSIS AND SYPHILIS OF THE CORD.[1]

BY F. X. DERCUM, M.D.,
OF PHILADELPHIA.
PROFESSOR OF NERVOUS AND MENTAL DISEASES, JEFFERSON MEDICAL COLLEGE;
NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

As is well known, there are certain symptom groups which roughly characterize both cerebral and spinal syphilis, and while striking departures from these symptom groups are not infrequent, these departures in no way militate against the truth of this statement. However, the subject must be viewed in the broadest possible light. A comprehensive view alone will assign to special symptom groups their proper value and prevent the undue exaggeration of their relative importance.

The following cases bear upon the interesting question of the relation of syphilis to spastic spinal paralysis, and also indirectly upon the question of Erb's form of spinal syphilis, and they are for this reason placed upon record:—

CASE I.—A. S., male, single, white, German, aged thirty-eight, baker by occupation, was admitted to the nervous wards of the Philadelphia Hospital, June 16, 1902, suffering from a weakness of the left arm and leg.

No family history of importance could be elicited. Both parents were dead. Patient had two brothers, but knew nothing of their medical history.

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[1] Read at the meeting of the American Neurological Association, September 15, 16 and 17, 1904.

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Personal History.—Had measles but no other diseases of childhood. Had gonorrhea at thirty-six years of age. Did not know whether he had had syphilis, but admitted frequent exposure.

History of Present Disease.—On Thursday, February 6, 1902, while at work he suddenly became dizzy. He had dimness of vision, and the right leg became weak—the leg, however, did not become paralyzed. He did not fall nor did he lose consciousness. He went to bed, and in two days had improved so much that he was able to get up and be about. On the following Sunday, however, he again suffered from an attack of dizziness. This attack was followed by loss of the power of speech, and for a time by inability to swallow; there was also marked weakness of the left arm, and to a less extent in the left leg. As before, he did not become unconscious. States that his mouth was drawn to the left. Loss of speech persisted for about three days and then entirely disappeared. Loss of power in the left arm and leg, however, has persisted up to the present time.

At the time of admission it was noted that he had some loss of power of the left arm and left leg. Both knee-jerks were plus, the right being slightly greater than the left. Ankle clonus and plantar clonus were elicited upon both sides. The Babinski sign was present upon both sides. The left biceps jerk was also plus. Some wasting was noted in the interosseous muscles of the left hand. The left leg also showed some atrophy. The pupils were equal, and reacted in a normal manner to light and accommodation; both were slightly dilated. The tongue deviated slightly to the right side. No sensory losses were noted. The general visceral examination was negative.

June 19, 1902, the following notes were made: The right leg and foot can be moved in all directions, with a good degree of strength. With the left leg all movements can be made, but not so well as with the right side; flexion and extension of the toes and foot are especially defective. The right arm can be moved with normal strength in all directions. The left arm is held with the forearm flexed upon the arm, and the fingers flexed upon the hand. The only movement the patient can make with this member is slight flexion of the forearm upon the arm. Slight rigidity of the left leg is noted when passive movement is made. Marked rigidity of the left arm is also present. The mouth is slightly drawn to the left, but when voluntary effort is made is moved better to the right than to the left. When the patient smiles no difference is noticed. The tongue is protruded slightly to the right; the patient can move the tongue from side to side, but cannot move it to the left as well as to the right; no fibrillary tremor is noted. There is apparently a weakened action of the pterygoids and masseters. There is no difficulty in chewing. There is no paralysis of the occipito-frontalis, nor of the corrugator supercilii. There is no ptosis. The patient seems to be unable to completely close the eyes on either side; forcible efforts to do so cause rapid tremor of both eyelids. The pupils are equal and react to light, but somewhat sluggishly; they react well to accommodation. The eyeballs can be moved well in all directions.

On the right side the plantar reflex is very active; the toes are first flexed and then extended. On the left side the plantar reflex does not seem to be as active as on the right; there is slow extension and separation of all the toes. The quadriceps jerk is increased, there is marked ankle clonus and slight patellar clonus on both sides. Both knee-jerks are plus, the left being the greater. The tendon jerks of the right arm are present and slight in degree. On the left side they are exceedingly active. A jaw jerk is also present. The cremasteric reflex is present, and is exceedingly active on the right side and rather sluggish on the left. The abdominal reflex is apparently a little more active on the right side than on the left.

The circumference of the left thigh is about a half inch less than that of the right at levels of eight and fourteen inches respectively below the anterior superior iliac spine. The muscles of the leg are more flabby than those of the right.

In both hands there is wasting of the adductor pollicis and interosseous muscles — more marked on the left side. The left arm, three inches below the olecranon process, is one inch less in circumference than the right arm at the same level. The left upper arm also measures an inch less than the right. There is also wasting of the left deltoid as compared with the right, and of the left supra- and infra-spinatus muscles as well as of the pectoral muscles. The myotatic irritability of the muscles of the left upper extremity is much increased. Fibrillary tremors are noted in the adductor pollicis and interossei muscles. There is no disorder of the sphincters. For three or four days the patient has had pain off and on over the right shoulder blade.

Speech is thick and indistinct; this he says is different from what it used to be. There are no sensory losses of the cutaneous surface; no loss of muscle sense; no loss of the sense of position. The feet and hands are cold; the left hand is slightly livid.

Reexamined on June 21, it is found that his station is very insecure, that he stands with both legs wide apart, that he is able to walk a few steps, but does so with great difficulty. The gait is not typically hemiplegic; the right leg is used comparatively well, but the left is pushed forward with some effort.

Reexamined on July 16, it is noticed that the right leg is somewhat weaker than before. Patellar clonus is present and marked upon both sides. Otherwise no change is noted.

An examination of the eye grounds by Dr. Crosky revealed contracted arteries, dilated veins and slight hyperemia of the left disc. There are no signs of neuritis, of atrophy or of extravasation.

Re-examined on the 15th of August, it is found that faradic contractility is somewhat diminished in the adductor pollicis and the interossei muscles of the left hand.

Examined October 16, it is found that there has been an appreciable change in his symptoms. It is found that while he can now flex and extend the right leg, the movements are awkward and distinctly ataxic. This is also true of the movements of the right arm. The left leg on the other hand is now distinctly spastic, and the movements of the limbs are decidedly restricted because of muscular rigidity. There is also marked rigidity of the left arm, the forearm and fingers being in a position like that of secondary contracture in hemiplegia. The left angle of the mouth is slightly depressed, the left side of the face is slightly flattened, while the left eyeball occasionally drops outward as though from weakness of the right internal rectus. The occipitofrontalis muscle apparently acts normally upon the right side; upon the left side its action is evidently diminished. The left brow is relatively smooth. The left zygomatic muscles are distinctly weak; the left angle of the mouth is not retracted as well as the right. As regards the trunk it is found that the left pectoralis major is firmly contracted, while the right is normal. The abdominal muscles and the muscles of the back of the trunk are apparently normal.

On October 17 the following note was made: While attempting to walk in the ward the patient suddenly became dizzy and fell, receiving a cut on his forehead above the right eye. Two stitches were required to approximate the edges of the wound. It was noted subsequently that his speech was not as good as before. There was also a slight rise of temperature and some general weakness.

On the 18th it was noted that the knee-jerks were equally pronounced upon both sides. Attempts at passive movements of the legs induced marked rigidity. Persistent ankle clonus was noted upon the right side, but not upon the left. A Babinski sign was now noted upon both sides. Sensory loss could be nowhere detected; tactile, thermal and pain sense were equally well preserved. There was no loss of the sense of position.

Eye examination by Dr. de Schweinitz, October 29.

Both discs are now distinctly degenerated, giving the appearance of a low grade interstitial neuritis. The veins are very full and the arteries contracted. The reaction of the pupils to light is normal, there is no diplopia. Rotation of the eye balls is normal; there is partial paralysis of both orbiculares palpabrarum, the patient being unable to completely close the eyes.

Re-examined on November 22, it is noted that since the last examination (made about a month ago) his condition has become much worse. His voice is weaker and more indistinct; he is now unable to remain out of bed; when sitting in a chair falls over. He makes no complaint. His face has a mask-like appearance; he is still unable to close the eyelids completely, and when he makes an effort to do so the palpebral fissure on each side remains open for from one-eighth to one-quarter of an inch. He has much difficulty in showing his teeth, and does it very imperfectly; cannot draw the corner of the mouth up on either side. The tongue deviates well to the right when protruded, and is unusually thin, but equally so on both sides and shows no fibrillary tremor. The pupils are equal, possibly the left is a little larger than the right. The irides react promptly to light. The movements of eyeballs are normal in all directions; no nystagmus.

Every now and then during the examination there are contractions of the facial muscles, such as occur in laughter, and this he does without having anything to amuse him.

He can move the right upper limb freely, but the movement is ataxic, and he has distinct difficulty in placing the first finger of the right hand on the end of the nose.

The left upper limb is paralyzed, and he can move it very slightly at the left elbow and in the left fingers. Contracture of the left elbow and fingers is very marked. Biceps tendon, triceps tendon, wrist reflexes in left upper limb much exaggerated. These reflexes in right upper limb slightly exaggerated. Sensation for pain is preserved in both upper limbs over the trunk and in the face; accurate studies of sensation can, however, no longer be made because of the mental condition. Has incontinence of urine and feces. The left lower limb is paralyzed; there is contraction in extension, and it is impossible to flex the left leg passively at the knee. The patellar reflex on the left side is exaggerated, but no movement can be obtained of the leg because of the contracture. The patellar reflex on the right side much exaggerated. Ankle clonus distinctly present on each side. Babinski reflex very marked on each side; the big toe is moved promptly upward. Sensation for pain preserved in the lower limbs.

December 26, 1902. Is unable to raise himself in bed. Can, however, turn over on his left side by grasping the rail of his bed with the right hand. Says he is comfortable and has no pain. Closes his eyes more incompletely than before; the palpebral fissure of the right side now remains open for about one-third of an inch, the left for about one-quarter inch. Has less power to show his teeth than before; both angles of the mouth can be feebly retracted, the left less than the right. When laughing the angles of the mouth are retracted much better and equally. The tongue is protruded only slightly and with difficulty; it deviates markedly to the right. There is no change in pupillary phenomena or ocular movements. The patient moves the right arm in various directions, but the movements are ataxic and jerky, the grip is very weak. Cannot move the left upper extremity except to make faint flexion movement at the elbow. There is no motion of the hand or fingers. Contracture of shoulder, elbow, wrist and fingers is present, as at previous examination. Reflexes, sensation and sphincters as before. He is able to move the right leg feebly in various directions, also the foot and toes. He is also able to move the left leg feebly, flexing it slightly at the knee. The left leg can now be flexed passively at the knee; it is not quite as rigid as at the last examination.

January 21, 1903. The patient has for the last twenty-four hours shown signs of failure, is unable to take nourishment and can only be roused with difficulty.

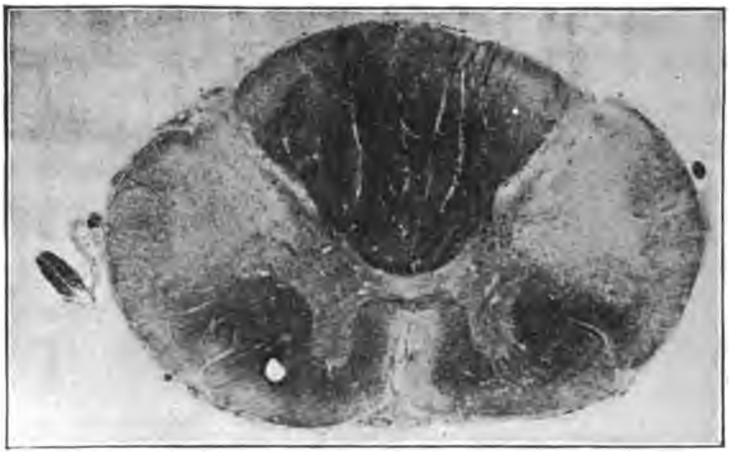

Fig. 1.—Transverse section of cervical cord in Case I., showing predominance of lesion in pyramidal tracts, direct and crossed.

January 22, 1903. Rise of temperature, 101.8 degrees.

January 23, 1903. Shows general and increasing failure. The pulse small and very rapid. The respirations are normal in frequency; the lungs are clear throughout. There is no distension of the abdomen. Temperature 100 degrees.

January 24, 1903. To-day the patient has shown very marked signs of weakening; cannot be aroused at all. The respirations are rapid, the pulse 180 and very small. It has not been possible to elicit a cause for his condition other than exhaustion. Rapid rise of temperature to 105 degrees; death.

Summary.—Sudden loss of power in right leg; sudden loss of power in left arm, and to a less extent in left leg. Rapid recovery of right leg; persistence of weakness in left arm and leg. Later recurrence of loss of power in right leg; involvement of right arm. At first no rigidity; later on rigidity established. Thenar and interosseous wasting in both hands, most evident in left; some wasting of left thigh. Tendon jerks plus; ankle clonus present; later Babinski sign on both sides. Bladder at first not involved, later on incontinence. No sensory losses. Gradual mental deterioration.

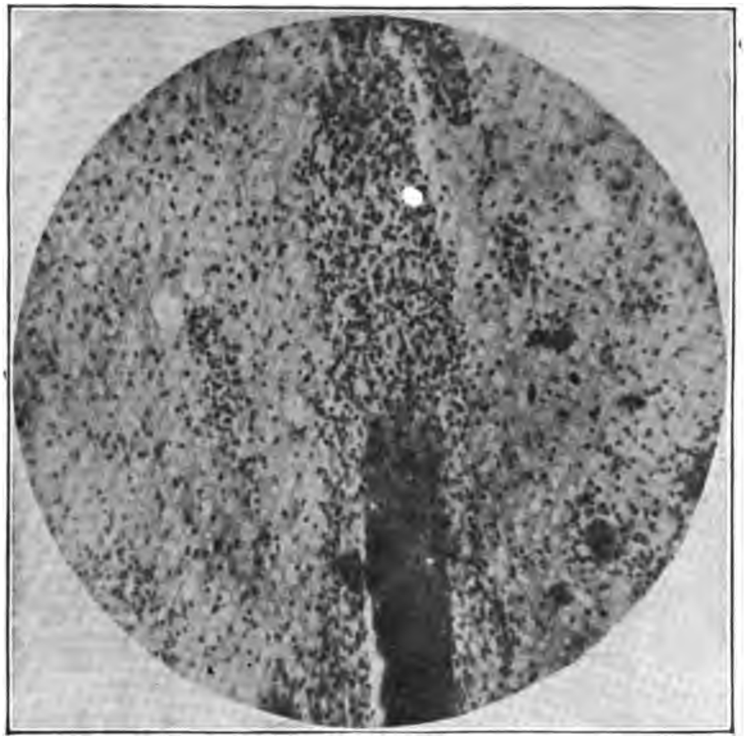

Fig. 2.—From Case I., showing inflammatory infiltration in pyramidal tract.

January 28, 1903—*Autopsy.*—This, unfortunately, had to be limited to the removal of the brain and cord.

Gross examination of the brain and cord revealed evidences of disease of the pia-arachnoid; opacities and thickening. No lesions whatever could be detected, either in the capsules or basal ganglia.

Microscopical Examination—Lumbar Cord.—Meningitis and vascular infiltration pronounced; moderate peripheral infiltration; marked infiltration and degeneration of the pyramidal tracts.

Dorsal Cord.—Meningitis and vascular thickening more pronounced. Moderate peripheral infiltration most marked anteriorly and laterally; hardly perceptible in posterior columns. Crossed pyramidal tract infiltration and degeneration very decided.

Cervical Cord.—Moderate meningitis and infiltration and thickening of vessel walls. Moderate general peripheral infiltration least marked in postero-lateral and postero-median columns. Very pronounced infiltration and degeneration in both crossed pyramidal tracts and in left direct pyramidal tract; slight degeneration in right direct pyramidal tract. The changes in the pyramidal tracts are greatly in excess of the other changes in the cord. The antero-lateral and antero-median groups of cells in the anterior cornua appear to be diminished in number upon both sides.

The degeneration of the motor tracts can be traced as far upward as the anterior pyramids, but not beyond; a small independent area of degeneration is noted in the inner third of the right crus, but the pyramidal fibers are intact upon both sides. The medulla and pons reveal meningeal and vascular involvement, moderate in amount. Similar changes are also noted in sections of the cortex, though they are here less pronounced than elsewhere.

Case II.—J. M., male, single, white, Austrian, age thirty-eight, sailor, was admitted to the nervous wards of the Philadelphia Hospital September 24, 1902. The memory of the patient is defective, and he is evidently somewhat confused. No family or personal history can be obtained. In reply to questions he admits alcoholism and veneral disease.

There is present decided paresis of the lower half of the left side of the face. The tongue is protruded in the median line. There is marked paresis of the left arm, with some rigidity at the elbow and shoulder and contracture and rigidity of the left wrist and fingers. The movements of the right upper extremity appear to be normal.

The right leg is almost completely paralyzed; the patient, however, can make faint movements with the toes. The left leg also presents a marked though less complete loss of power; the patient can elevate it from the bed but cannot hold it up long. He moves the toes of this foot freely. There is present also incontinence of the bladder and bowels.

The jaw jerk is slightly increased. The right biceps and triceps jerks are increased. The right wrist jerk is present and pronounced. The right knee jerk is likewise present and not exaggerated. There is present a right disappearing ankle clonus. The Babinski sign is also present upon the right side. The left biceps and triceps jerks are much increased, as is also the left wrist jerk. The left knee-jerk is present and decidedely exaggerated. There is present a faint left ankle clonus. The Babinski sign is also pronounced and decided upon the left side. A tendon Achilles jerk is present upon both sides. Cremasteric and abdominal reflexes are present but sluggish. There is no loss of sensation. There is no pain or tenderness along any of the nerve trunks, nor are there any vasomotor symptoms. The speech is somewhat long drawn and indistinct. The tongue is deviated slightly to the left and tremulous. There is no involvement of phonation. The patient apparently does not hear as well with the left ear as with the right, but there is no decided deafness.

The pupils are unequal; the left being the larger. Both pupils fail to react to light. There is no paralysis of the external ocular muscles.

The patient complains of pain in the right chest. There are present bronchial breathing and moist rales. Heart sounds are dull and distant. The pulse is slow and weak, though regular.

Examined October 18, 1892. The left knee-jerk as before is exaggerated. There is present a faint left ankle clonus. The right knee-jerk continues to be normal. A Babinski reflex is present upon both sides, but not quite as marked as at the previous examination. The right arm is flaccid, the right leg is flaccid. The left arm is less rigid than upon admission, though decided resistence to movements is still present at the elbow. The wrist and fingers are less rigid than at the first examination, Otherwise there is no decided change. The patient is evidently growing weaker. There is a slight rise of temperature. Pulse is rapid, while the breathing is stertorous. The patient is now quite stuporous.

The examination of the urine reveals the presence of some hyalin and a few granular casts with a small quantity of albumin.

Death occurred October 20.

Summary.—Paresis of left arm, very marked paralysis of right leg, paralysis of left leg. Tendon jerks of arms, especially of left, exaggerated. Right knee-jerk not exaggerated, left knee-jerk plus. Faint left ankle clonus. Disappearing right ankle clonus; Babinski sign upon both sides. Rigidity, later flaccidity. No sensory losses. Incontinence.

Autopsy.—The visceral changes noted were chronic pleurisy and a pronounced nephritis. There was also a syphilitic scar upon the liver.

The spinal dura appeared to be adherent to the anterior surface of the bony canal. This was most marked in the lumbar region. The pia mater of the brain was thickened and infiltrated upon either side of the longitudinal fissure and over the vertex generally. Otherwise the brain presented no striking peculiarities upon gross examination.

The skull presented over the occipital protuberance an extensive periostitis with some roughening of the bone. In the left frontal region also there was a slight depression of the external table.

Microscopical Examination—Lumbar Cord.—Marked meningitis and thickening of vessels. Marked infiltration of the pyramidal tracts; moderate infiltration of postero-median columns, slight general peripheral infiltration.

Cervical Cord.—Marked thickening of pia and infiltration of vessel walls. Moderate general peripheral infiltration. Marked infiltration of right crossed pyramidal tract and of left direct pyramidal tract. Infiltration of left crossed pyramidal tract, less marked than in its fellow. Faint involvement of right direct pyramidal tract. Slight involvement of direct cerebellar tract upon either side; faint involvement of posterior columns. The changes predominate greatly in the pyramidal tracts.

Case III.—P. N., single, white, German, hatter by occupation, age sixty-six. Was admitted to the Philadelphia Hospital on September 20, 1901.

Complains of pains in the legs, right side of the body and of difficulty in walking. Mental condition poor.

No satisfactory family or personal history can be elicited. Alcoholism and venereal diseases admitted.

It appears that the present trouble began some two years ago with pains in the legs and thighs, and that it has gradually grown worse. He first noticed numbness and stiffness in the legs about a year ago, but this only lasted for a short time. It came back at intervals, and four weeks ago he was obliged to use a cane in walking. About a week previous to admission was obliged to go to bed, being unable to walk because of pains and jerking in his legs.

Present Condition.—His general appearance is that of a well developed but rather poorly nourished man. The ninth, tenth and eleventh dorsal vertebræ are unusually prominent, and he complains of pain when pressure is made over them. He says at times that he has pain in this region when walking. The patient is able to make all movements with his legs, but the movements are weak. There is no rigidity, the muscles are flaccid, and there is no atrophy. The knee-jerks are increased upon both sides. Ankle clonus is absent upon both sides. Babinski sign is present and marked upon both sides. Achilles jerk is absent. There is a fair degree of strength in both upper extremities. They are apparently not involved. There is no involvement of the muscles of the face or of the tongue. The extraocular movements are well performed. The pupils are unequal, the right being contracted and yielding no response either to light or accommodation. The left pupil on the other hand is apparently normal, and responds to light and accommodation. The patient says that his arms have grown somewhat smaller lately. No marked atrophy of any special muscle or groups of muscles can be detected; however, the infra- and supra-spinatus muscles seem to be somewhat wasted, as do also the latissimi dorsi. Fine fibrillary tremors are noted in all of these muscles. There is a coarse intention tremor of both hands.

The patient complains of a sharp lancinating pain, extending down the legs. The pains are worse toward evening. The patient states that the pains are attended by spasm of the muscles, which cause him to kick his feet about. He also complains of delay in micturition. No anomalies of sensation can be detected. The speech is rather slow and deliberate, but not really abnormal.

Reexamined May 6, 1902, it is noted that the patient has a slow and slightly spastic gait. There is no involvement of the upper extremities. An intention tremor is noted as before. The knee-jerks are both much exaggerated. A patellar clonus is present upon both sides. The Babinski sign is present and pronounced as at the previous examination. An Achilles jerk is also present and pronounced. No ankle clonus is present upon either side. Pain is elicted upon pressure over the lower dorsal and upper lumbar spine. The possible deformity noted at the previous examination cannot be confirmed. Sensation as before is normal. The wasting of the infra- and supra-spinatus muscles noted at previous examination, is evident as before. The movements of the arms are performed normally. The biceps jerk is now decidedly increased on both sides.

Reexamined on December 11, 1902. There is no facial palsy or asymmetry. The tongue seems small and is protruded with some difficulty. The arms now reveal marked wasting. In the right arm there is a general wasting of the upper arm and of the forearm. It is more marked in the flexor groups than in the extensors. There is also decided wasting of the thenar and hypothenar eminences, with a less marked wasting of the interosseous muscles. Bicipital, tricipital and wrist jerks are present and exaggerated. In the left arm there is a wasting of the upper arm, not quite as marked as in the right. There is some wasting of the muscles of the forearm, especially in the flexors. The palm of the hand is decidedly hollow; there is some wasting of the thenar eminence and a less marked wasting of the hypothenar eminence. The reflexes are plus as in the right arm.

In the right lower limb the thigh is decidedly wasted, the adductor group being somewhat better preserved than the other muscles. There is a general wasting of the lower leg, though this is not excessive. In the left lower limb the thigh is wasted in a similar manner as the right, and the wasting is more pronounced. The left leg, in keeping with this, is somewhat more wasted than its fellow. The reflexes continue as at previous examination.

The pupils are unequal, the left being the larger. The latter does not respond to light, while the right reacts in a normal manner.

An examination of the urine reveals pus cells, granular casts and albumin and a specific gravity of 1020.

The man presented no visceral signs of moment or any notable changes of temperature until May 26, when it rose to 100 degrees, then fell to normal and continued so until November 28, when it rose to 100.4 degrees. It subsequently again fell to normal until the afternoon of the 30th, when it rose to 102 degrees, and remained at this figure during the night. On the morning of January 1, 1903, it fell back to normal, and continued so until the morning of the 3d, when it rose to 101.4 degrees, and then to 104.2 degrees, and subsequently fell back again to normal until the 7th instant, when it rose again to 104.2 degrees, and remained high until the evening of the 8th. Subsequently it fell again, and fluctuated from 100 to 102 until death set in. Death finally occurred as a result of nephritis on the 11th of January, 1903.

Summary.—Pains in legs, pain referred to lower dorsal spine; weakness of both legs. Gait spastic. Knee-jerks plus. Babinski sign upon both sides. Upper extremities not involved. No sensory losses. Delayed micturition. Later some atrophic changes in arms and legs.

Autopsy.—The principle changes noted were marked degenerative changes, arteriosclerosis, chronic endocarditis and chronic nephritis. There was also a hypostatic pneumonia. The brain and spinal cord revealed little change to gross examination.

Microscopical Examination.—Marked meningitis and thickening of vessel walls. General peripheral infiltration and degeneration, especially in the pyramidal tracts of either side; less marked changes are noted in the postero-median and postero-lateral columns.

Cervical Cord.—Moderate thickening and infiltration of the pia and of the vessel walls. Slight general peripheral infiltration, a little more evident on the lateral and posterior aspects of the cord.

In all of the above cases the lesion conformed in general terms to that most commonly found in syphilis, namely, a meningomyelitis. It will be noted, however, that in Case I, the meningitis was only moderate in degree, relatively slight in the cervical region, but marked in the lumbar region; while in Cases II and III the involvement of the membranes and vessels was everywhere pronounced. It is noteworthy further that in all of the cases there was a decided involvement of the pyramidal tracts, which involvement was far in excess of the involvement of any of the other tracts. This excess of pyramidal tract disease was especially striking in Case I (see Figs. 1 and 2); though noticeable in Case III, the difference between the pyramidal and general peripheral involvement was not as marked. It would seem almost as though some selective action had taken place in favor of disease of the pyramidal tracts. The lesion in each instance was inflammatory in character, and was evidently secondary to a preexisting syphilitic disease of the membranes and vessels.

It will be recalled also that in Cases I and II atrophic changes in the muscles had begun, which strongly resembled those seen in amyotrophic lateral sclerosis. That spinal syphilis now and then simulates amyotrophic lateral sclerosis is, of course, well known. In the present instance the atrophic changes are probably to be attributed to involvement of the motor root fibers rather than to involvement of the anterior cornua, though in Case I there seems to have been some diminution in the number of cells in the anterior cornua in the antero-median group in the cervical region.

Cases I and II, save in the presence of a spastic paralysis, in no way resemble the form of spinal syphilis known as Erb's syphilitic spinal paralysis. Case III, however, presents clinically a distinct resemblance to Erb's form, though it cannot be regarded as a typical instance. Erb's form, it will be remembered, is a symptom group in which the patient presents spastic paresis of the lower extremities, spastic gait, moderate loss of power, moderate or no rigidity of the affected muscles, exaggerated tendon reflexes, disturbances of sensation slight or absent, and weakness of the bladder. The upper extremities, the brain, cranial nerves and the pupils present no symptoms. However, as Nonne has pointed out, Erb's symptom-group embraces a series of most divergent cases. Thus there are on the one hand cases which present an almost pure picture of spastic spinal paralysis, *i. e.,* cases in which there is spastic paresis of the lower extremities of slow development, and with slight contractures, changes of sensation exceedingly slight or absent, and almost completely normal bladder and sexual functions. On the other hand, there are cases in which a motor paraplegia appears, relatively rapidly, and in which there are objective sensory changes, decided impairment of sexual power and of the function of the bladder; later there may be also a secondary cystitis and even decubitus. The time of appearance of the symptoms also varies greatly—for instance, from three to twenty years. Erb, as late as 1902, in his address before the Post-Graduate College in London, expressed the opinion that the symptom group described by him under the term syphilitic spastic paralysis. constituted a system disease. He cited four autopsies—namely, one by Westphal, one by Eberle, one by Nonne and one by Williamson—in support of his position. The findings were practically those of a combined system disease, that is, involvement of the pyramidal and direct cerebellar tracts, with a less involvement of posterior tracts—sometimes of the columns of Goll and sometimes the columns of Burdach as well. Nonne has also placed upon record a case which revealed a myelitic degeneration from the eighth to the eleventh dorsal segment, but in addition a combined system disease, above and below. Other cases, such as those of Strümpell, Williamson, Long and Wiki, are also cited by Erb. They were likewise cases in which the picture was not exclusively one of combined system disease, but also of more or less marked degeneration in the dorsal cord, so that it was difficult to determine how much was primary degeneration and how much was secondary. Nonne, after a review of the pathological changes in Erb's symptom-group, sums up the possible anatomical findings as follows: First, a chronic transverse myelitis with ascending and descending degeneration; second, a chronic transverse myelitis with a primary degeneration of the pyramidal tracts; third, a chronic transverse myelitis alone, which is either accompanied or unaccompanied by disease of the vessels of the spinal cord.

While the existence of Erb's symptom-group of syphilitic spastic spinal paralysis must unquestionably be conceded—for every nervous clinic and every large nervous hospital ward presents a number of such cases—it must be admitted on the other hand that a corresponding and constant anatomical lesion has not been found. Erb is inclined to consider the affection as a post-syphilitic toxic disease, analogous to locomotor ataxia, but in view first of the ill-defined limitations of the symptom-group itself and of the admittedly variable character of the post-mortem findings, we cannot but regard this position with doubt. Indeed, we are inclined to agree with Oppenheim, Leyden and Goldscheider, Bruns, Reiner, Orlowsky and Korniloff, and Marie, who regard Erb's form as merely a stage of syphilitic meningo-myelitis, and who see in it nothing more than a transverse syphilitic myelitis. That an examination of the cord in late cases of this form of disease may, as Oppenheim has pointed out, reveal but little change in the membranes, goes without saying; while at the same time the changes in the columns may be pronounced and permanent. Furthermore, to suppose that the post-syphilitic toxin would in one instance give rise to tabes dorsalis, and in another to an entirely different disease, introduces a new difficulty. The one spinal disease of post-syphilitic, or para-syphilitic origin, is beyond all question tabes; it is the posterior columns upon which the suppositious toxin expends its energy. Again, the evidence at hand is overwhelming to the fact that syphilis in the pretoxic stage, that is, during its activity as an infectious process, invades the cord through the membranes and vessels. It is true that the membranes and vessels may be only slightly involved, or, perhaps, as is claimed, escape, while the cord itself becomes the seat of specific infiltration. Further, it would almost seem that syphilis during the infectious stage affects by preference the motor pathways, *i. e.*, the pyramidal tracts, not only the crossed pyramidal tracts but the direct pyramidal tracts as well, together with possible ascending and descending degenerations. How this tendency to excessive involvement of the pyramidal tracts arises, must for the present remain a matter of speculation. That it is a distinct element in syphilitic spinal paralysis the evidence appears most clearly to show, and that it is not dependent upon mere peculiarities of blood supply or relative anatomical position of the tracts within the cord is proven by the fact that the same predominence of lateral tract involvement is not noted in other forms of infectious myelitis.

A PRELIMINARY REPORT OF THE POSSIBLE RELATIONSHIP OF NEURO-FIBRILLAR CHANGES TO INSANITY.[1]

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As Nissl states, it is necessary in order to have a pathology of the nerve cell that there be a well defined anatomy of the cell. The neurologist is at present engaged in studying the relationship of cells and the tracts of their processes.

To the psychiater it would seem that the morphology and physiology of the individual cell were the principal things to be studied; for it is from the structure and function of the nerve cell and its processes that we hope to learn something of the relation of mind to the physical process.

The value of becoming conversant with the morphological relations existing inside the nerve cells is without question, when we think of being able to trace a bearing on function in the capacity they exhibit for responding to external irritation and of storing up and reproducing happenings which go on outside of the cell.

During the past summer, in the laboratory of Friends' Asylum, we have been studying changes in neuro-fibrils as shown by the method of Cajal, published in the *Compt. rend, de la Soc. de Biol.*, Dec. 18, 1903, with a view of determining the possible relationship of changes in fibrils to insanity.

As far back as 1844 Remak[2] called attention to a fibrillary structure in certain axis cylinders and nerve cells.

Max Schultze followed with notable researches, in which he demonstrated that fibrils are seen in all parts of the cell and also in the dendrites as well as in the axone.

He said that the nucleus lay imbedded in the finely granular fibrillated material, but did not have any connection with the fibrils.

Then, in 1885, Nissl described the granular bodies which bear his name, which Rosin thinks should be considered in their chemical nature rather than morphologically; Benda having seen this same appearance in glands and Cajal in leucocytes and connective tissue. Macallum showed that there was iron in these bodies. While Haliburton distinguished a nucleo-albumin containing phosphorus; and from the quantity of work which has been done by investigators we must accept the suggestion that they are an index to the internal metabolism of the cell. These observations and the chemical experiments of Held and others tend to throw the burden of evidence of conduction on the achromatic substance.

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[1] Read at the meeting of the Philadelphia Neurological Society, October 25. 1904.

[2] Barker, "The Nervous System."

The majority of investigators also seem to be agreed that the achromatic or non-stainable portion of the cell body is closely allied, if not identical, with that of the axone. Certainly the fibrils are in the non-stainable portion (in the sense of Nissl) of the cell.

Apáthy in describing his nerve cell states that it produces neurofibrillæ just as a muscle cell produces muscle fibrillæ. He also compares nerve cells with muscle cells in their histogenesis, and believes that a nerve cell is no more capable of the highest functions of conduction before the appearance of the neurofibrillæ than is a muscle cell of the highest functions of contraction before its peculiar fibrillæ have been differentiated. The stain of Apáthy, however, has not been of value when applied to the nervous system of higher mammals. Apáthy claims that the neuro-fibrils in the lower animals represent the essential element of conduction. Bethe[3] shares this view.

Held looks upon the ground substance as a whole, as a means of propagation of stimuli.

Leydig[4] attributes conduction to the plasma which fills the meshes of the cellular network.

Others say it is the spongioplasm.

Butschli considers the frame work of the nerve cell protoplasm the histological basis of conduction. The opinions on these points are various.

Considering the fibrils as demonstrated in the peripheral nerves by Bethe's method with osmic acid, he states that where the nerve has been well stretched we see the fibrils in straight lines and individually separated, lying side by side, without any connection at all, until they bifurcate to branch off, and that they never end except when cut by the knife. Consequently he regards them as real individual fibrillæ, and of circular form. He further says that there is no difference between the neuro-fibrillæ of the efferent and afferent fibers; and that when in the Ranvier's nodes, the fibrils are kept at certain distances from each other, and this can be explained as an insulating function, the insulating substance preventing the contact of the threads.

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[3] Bethe, Alleg. Anat. und Phy. des "Nervensystem."

[4] Leydig, Archi. für Anat. and Physio., 1897.

After many experiments of pressure on the nerves he arrived at the conclusion that the perifibrillar substance is completely interrupted in the Ranvier's nodes, and only the neuro-fibrillæ pass continuously and uninterruptedly from one medullary partition to another. This being the case the fibrillæ are the conductors of the nerves.

In 1896 Lugaro[5] formulated an important generalization with regard to the respective significance of lesions of the chromatic and achromatic parts of the cytoplasm. He maintained that alterations to the chromatic parts do not represent more than a reaction of the cell to a disturbing force and are reparable; while on the other hand, alterations of the achromatic parts are to be regarded as degenerative and irreparable. From experimental pyrexia and poisoning, some observers are led to maintain that chromatolysis has little significance as a pathological change; for cells showing many changes had still been able to perform their functions, and it has thus been argued that the Nissl bodies can have no marked importance in functional activity. Lugaro, however, holds that while the activity does continue, yet it is less in degree, and that the chromatic part fulfils its functions through its chemical and not its morphological structure. He thinks the morphological conditions necessary to function consist in the structure of the achromatic substance, and the intensity of function depends on the chromatic.

If this view be true there could be considerable mental weakening due to chromatic chemical changes before there would be noted any marked change in achromatic substance.

In poisoning by ether or chloroform there may be no demonstrable lesion, while, even after functional disturbances have disappeared after compression of the abdominal aorta, the lesion of the chromatic parts is present.

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[5] Lugaro, Riv. di patol. nerv. e ment, 1896 and 1898.

But we can hardly doubt that the chromatic part plays a very important rôle in the functional metabolism of the nervous element and that its alterations are an index of a nutritive change.

Marinesco and Robertson[6] agree that yellow pigment is an evidence of senile involution, and with the formation of pigment there is usually an associated destruction of spongioplasm and trophoplasm, which are destroyed at the seat of the granular degeneration.

The granular disintegrations of the chromatic particles, that is, the breaking up of the aggregation of granules that form the Nissl bodies and the gradual disappearance of the individual bodies themselves, occurs even in non-nervous diseases, and is commonly attributed to pyrexia, terminal autointoxication, or local vascular lesions. In nervous disease it is usually accompanied by graver changes, such as we find in the fibrillæ.

The great majority of authorities are agreed that the fibrillar portion of the achromatic substance subserves the function of conduction of the nervous wave.

The non-organized portion of the achromatic substance is believed by Marinesco to be the seat of intense chemical phenomena, and of such importance for the nervous element as to be appropriately designated trophoplasm. That this substance is the seat of important metabolic changes is amply confirmed by other observers, especially by G. Levi, from study of fuchsinophile granules.

Golgi has stained an endocellular network which he thinks is a partial manifestation of finer and more complex structures. He has also described a reticular investment, thought by Bethe to be fibers impinging on nerve cells.

Held describes a fine meshed network which he says is not fibrils but a framework, and speaks of fine granules (neurosomes) at the nodal points of the network. These neurosomes converge at the axone hillock in lines, and in Held's pictures they have the appearance of a Cajal fibrillar structure, and might be granules lacking cement substance to form them into fibrils. In many sections stained with Cajal's method one gets the appearance of the fibrils having a granular makeup in healthy conditions, which is due to an imperfect impregnation.

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[6] Robertson, "Pathology of Mental Diseases."

The pathology of insanity has been recently discussed and has been declared insufficient. The most recent contribution is that of a case of dementia præcox,[7] which would indicate that the status of knowledge is as follows:—

"Pale yellow pigment is seen in majority of cells, central chromolysis is frequent. The cell is often swollen. The nuclei are atrophied, swollen, displaced and distorted. An endonucleolus is occasionally present. Folding of the nuclear membrane is rare. Neurophagocytosis is occasionally seen. The glia nuclei are slightly increased about the cells and occasionally cover them. Fragmentation of the cells is rare. The small pyramidal cells are frequently seen twisted upon their axes."

In the cerebellum all cells showed a granular appearance. Sections stained with Bethe's method showed the intracellular fibers distinctly stained except in the central part, where there was a granular appearance, and in some cases this involved nearly the whole cell.

The author seems to think tangential fibers are decreased in number, and that there is some increase in neuroglia tissue.

In order to advance the theory that insanity is particularly related to neurofibrils it was necessary, on account of our inability to get but two brains of insane persons at autopsy to approach it with the anatomical and physiological features bearing on the subject and the results of experiments on animals.

We found in our work at Friends' Asylum with fresh normal tissue from a rabbit by Cajal's method, large-sized cells in the medulla with fibrils running in through a process, then along the periphery of the cell, and going out by another process not necessarily an axone. Other fibrils approach nearer the nucleus and give off collaterals and continue on and out another process. Some advance directly to the nucleus, and apparently end in a network.

These features were especially noted in a large cell in the cord of a rat, where around the nucleus, there was a reticulum, while in the periphery were larger fibrillæ placed more or less parallel.—(Plate I, Fig. 1.)

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[7] Dunton, Journal of Insanity, April, 1904.

In the cortex the cells are of several sizes, some are small with a nucleus, and then a layer of fibrils around it which depart from the cell by several processes.

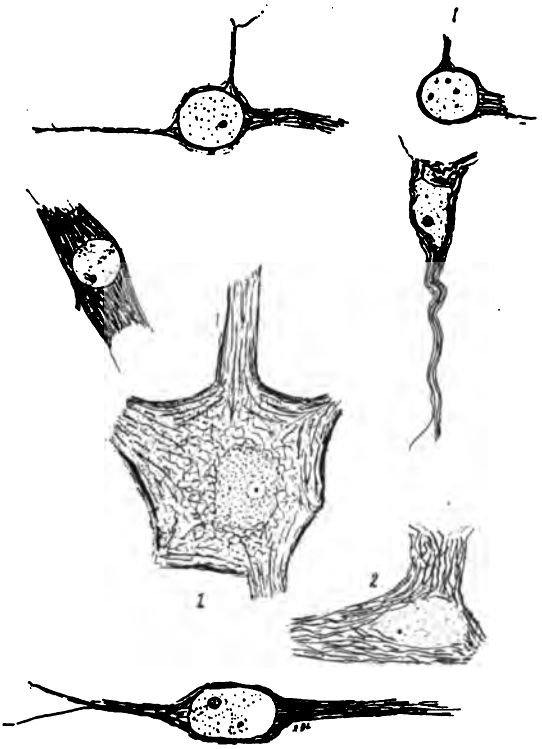

Plate I.—Normal cells from a rabbit and a rat; Cajal stain; 1. Cord of rat; 2. Medulla of rabbit.

These fibrils seem to run in one process and out another, it is difficult to see any collaterals given off. (See Plate I.)

The fibrils in the cord and medulla are larger and coarser than those in the cortex of the rabbit. In the larger sized cells in the cortex the cell body is broad, the fibers run more or less parallel, as is shown in the drawing, running from the processes at one extremity to the processes at the other. When the fibrils enter a process they are placed closer together, and give a cord-like appearance.

The nuclei in all the cells seem to be alike in many ways; there are small sized granules closely packed in some parts, leaving vacant spaces at other parts. The granules color yellow.

The nucleolus stains darker, a reddish yellow, and in it are dark granules moderately close together.

Some cells show spaces between fibrils in such an arrangement as to envelope Nissl bodies. Nissl, when speaking of bodies stained by his method and the non-staining portion, suggested the presence of fibrils in the non-staining part.

In a good stain the sections should have a reddish-brown background; if the pieces are above an eighth of an inch in size, the sections show pale centers, while the part correctly stained will be midway between that and the periphery, which is usually too dark to obtain good pictures.

In a good section the intracellular fibers take on a blackish color, but they do not stain as completely as they do in Bielschowsky's stain. The cell bodies should be a shade darker than the surrounding tissue and the fibrillæ a blackish color. The best results were obtained with a two per cent. silver nitrate solution, kept at 35 degrees C. for six days, washed in water two minutes, and then placed in one per cent. pyrogallic acid twenty-four hours, washed in water two minutes, alcohol ninety-five per cent., alcohol absolute, xylol, paraffin. Cut sections at two microns. When the sections are pale, if bathed for ten minutes in the following solution they improve:—

Amm. sulphocyanide..................	3.
Sod. hyposulphite.....................	3
Aqua	100.

Before using add a few drops of a one per cent. gold chloride solution, wash in water and clear. Many sections are not satisfactory, and it is necessary to go over a considerable number to get good ones, but when they are good they are apt to be so throughout.

In a normal brain, examined thirty-three hours after death, certain changes in the fibrils were noted, probably due to postmortem changes; differences which would not be noted in our ordinary stains. The fibrils in the majority of cells, in the region of the nucleus, had undergone granular degeneration. In the processes could be seen a few straggling fibrils intact, but these had degenerated on reaching the cell body; in some cases becoming flat and transparent. (See Plate II, Fig. 2.)

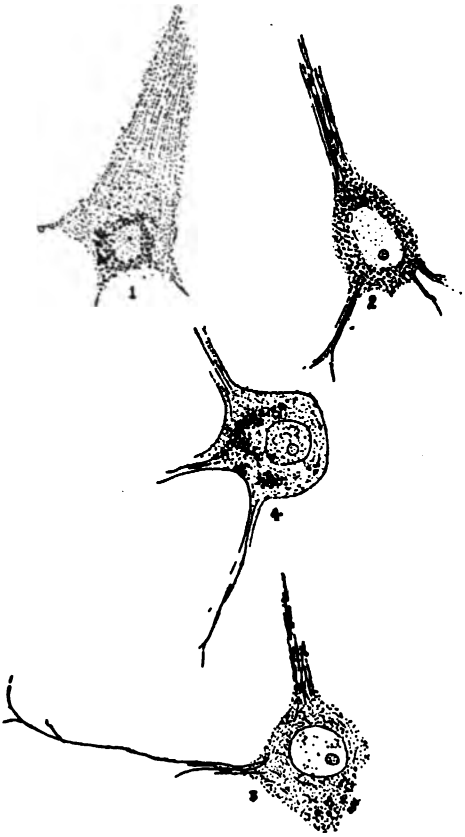

Plate. II.—Changes seen in cells in: 1. Paresis; 2. 33 hours post-mortem; 3. Starvation; 4. Insanity.

To create conditions which might simulate insanity, a rat was exhausted by starvation. His energy disappeared, and he sat in a corner of the cage perfectly still, he paid no attention to the rattling of paper or to the poking of a stick. On stirring him he seemed barely able to move. He was killed mechanically at the end of four and a half days. In the medulla were noted cells quite normal in appearance, and also cells showing distinct granular degeneration of the fibrils. At one place there were two cells side by side, one normal, and the other showing no healthy fibers at all. In the cord was a similar picking out of cells, which degenerated and a leaving of normal cells. In some cells where the fibrils show distinctly; there is a difference in their size, some being fine and interlacing, others course. In the cortex (Plate II, Fig. 3) many cells are entirely granular, often including the nucleus, which is filled with coarse granules, staining darker than the normal nuclear granulation. In the cell body are areas of a coarse black pigment. The fibrils in most of the affected cells can be demonstrated in the processes, while the cell body shows short broken pieces and fine granular lines. The nucleolus can be demonstrated, and in it a few granules, scarcely ever more than six in number.

In the second experiment an attempt was made to exhaust a rat by continuous running in a moving squirrel wheel. At the end of two and one-quarter hours the animal seemed to be quite exhausted.

Sections, however, showed only the beginning change, that is, taking the stain faintly and showing a reddish tint with some enlargement of fibrils.

In the case of insanity following we find practically the same changes as seen in the starved rat, but differing from the post-mortem change by the addition of pigment.

The case, sixty-seven years old, was one of manic-depressive type of long standing, which had developed into a very mild dementia. The patient had been able to be around, go to church, and to do sewing; she had been quite active, and had had no serious attack for five years. Sixteen days before death she became melancholic and nervous. She lay in bed, in restless motion, face pale and tense, pulse irregular and intermittent, feet and hands cold. She would lie muttering to herself and evidently in great distress, but could not be induced to speak. She paid little or no attention when spoken to, and resisted everything done for her. One morning she arose, moved a short distance, returned, and fell on the bed, dead.

Autopsy showed marked myocarditis. In this case we do not see in the brain cells the lesion of the mild dementia, but do see the cause of the acute profound depression coming on top of it. The poor circulation probably caused an anemic condition of the brain, which affected the neurofibrils, and from that, we would argue, followed the depression.

The fibrillæ are almost absent in the cell bodies, the remains can be seen often as lines of fine granules, traversing a field of fine granulation. In the cellular processes they are less affected, but are not intact, and stain less readily than the normal. Many cells contain a large amount of black pigment (Plate II, Fig. 4).

Thionin shows pigment, vacuoles and fine granulation, but no signs of fibrils at any point.

The nuclei are partially filled with corpuscular bodies, staining yellow. The nucleoli are very occasionally crumpled, but usually show from three to seven dark granules within.

The small nerve cells show similar lesions of broken fibrils and granulations.

The cord and medulla show the same changes as the cortex, which would incline one to the view of anemia as a factor in the degeneration and the primary cause of the depression.

Tissue which has been hardened in formalin gives fairly satisfactory results with one per cent hydrochinone has been used instead of one per cent pyrogallic acid.

Some pieces of cortex, so hardened from a case dying of paresis, showed the absence of fibrils in the cell body with the remnants of them in the processes; also coarse granulation in the nucleus and pigment in the cell body. (Plate II, Fig. 1).

Our studies and experiments thus far at Frankford would seem to prove the intimate relation which the neuro-fibrils of nerve cells bear to mental processes.

In the experiment of the partially exhausted rat was found the earliest change, that of swollen fibrils and inability to take the stain deeply. In the rat affected by starvation we have another phase of the etiological factor in the change of the neuro-fibrils, which plays an important part in the insanities arising from mal nutrition. We have under way other experiments to study the effects of morphia, cocaine, etc., so often a cause of insanity.

The changes quickly wrought within thirty-six hours postmortem tend to show the delicacy of the structure of the neurofibrils.

The case of depression so quickly following a change in circulation which affected markedly the neuro-fibrils, would seem to indicate a close relationship between fibrils and mental manifestations. The absence of fibrils in complete paretic dementia would be what would be expected if such a relationship is true.

If we advocate an hypothesis, it is distinctly such until it is proven a fact. The establishment of relations, whether structural or functional, to be of value should be constant; and if by studying insanity pathologically, we should find these changes in a large number of cases it would then be possible to group psychoses more scientifically and to have a definite pathology of the various forms of disease. Finally, if this should prove true, undoubtedly to Cajal belongs the credit of demonstrating the method by which it can be done, and to Marinesco[8] great praise for the brilliant elucidation of his numerous experiments.

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[8] Revue Neurologique, May 15, 1904, and August 15, 1904.

A CASE OF TUMOR OF THE OCCIPITAL LOBE.[1]

By Philip Zenner, A.M., M.D.,
of Cincinnati.

The following case of tumor of the brain is reported because its clinical history is one of unusual interest.

Mr. A., aged forty-three. Patient comes of a gifted but eccentric family. His father is said to be mentally unsound, and to have been so for many years. Patient himself is a brilliant man, with some tendencies to eccentricity. He has occupied all positions on a newspaper, and now has been a railroad man for many years. He has been the manager of many men and affairs, and thereby accustomed to be very active and busy. He has been a heavy smoker for years, and also has been addicted to the use of considerable whisky daily. He denies syphilis, but his wife has had several miscarriages. The history of headache in his case is not clear; and its causes, considering his habits and all, are, also, in doubt. It seems probable that the last four or five years headaches were common, but that they were worse the past year or two, during which time he was accustomed to take one or several headache powders daily. A powder usually relieved his headache at once, so that he could continue his work without interruption. In this way he got into the habit of taking a powder whenever he felt a headache coming on, and, very likely, rarely a day passed that he did not take several.

As before stated, the nature of these headaches is not altogether clear; that is, how far they were due to his habits, how far to a growing neoplasm.

His last illness appears to have begun abruptly February 11, 1903. On that day as he got off of a street car he ran into a post on his right side which he did not see. From like experiences it soon became apparent that he did not see on the right side. It is quite probable that the loss of vision came on suddenly on the date mentioned. An examination by Dr. C. R. Holmes on the following day revealed an almost complete right hemianopsia. The left half of the field of vision was normal in outline. Vision in the left eye was 20—20. In the right eye there was an old amblyopia, vision 20—200. Disks were normal. From this time patient suffered with severe and continuous headache. There was also, especially at times, some slowness of thought and difficulty in finding words. One night there was such a degree of mental confusion that the patient did not know where he was.

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[1] Read by title at the meeting of the American Neurological Association, September 15, 16 and 17, 1904.

On February 26, 1903, Dr. Holmes found the disks cloudy at the margins and congested, the condition being more marked in the right eye.

I first saw the patient March 3, 1903, in consultation with Dr. A. Schwagmeyer. He was in bed and suffering severely with headache, which was chiefly on the left side, and frontal. He thought the pain had been chiefly on the left side for several days, but was uncertain just where it had been prior to that time. But both at this time and subsequently there was a certain indefiniteness as to the location of pain. His answers were rather vague; often he would not pretend to localize the pain. His mind appeared clear. He answered questions readily, had no difficulty in naming objects, and expressed himself well. The right hemianopsia was very apparent, and there was at this time well marked double optic neuritis. Otherwise there was no paralytic manifestations. The pupils responded to light, and responded equally well whether the light was thrown in from the right side or the left (Wernicke's pupillary response). There was no motor paralysis or ataxia anywhere. All kinds of sensation, including stereognosis, were normal. The knee-jerks and plantar reflexes were normal. The patient could read, and wrote his name fairly well. His pulse was only 60. His sleep was very poor on account of pain, and his appetite and digestion were not good.

My diagnosis was tumor in the left occipital lobe. Though he gave no history of syphilis, he had been receiving inunctions of mercury for about a week. After discontinuing this for some days we began giving mercury hypodermatically, and subsequently iodide also. The latter was given but a short time on account of the condition of the stomach.

From the date of my first visit I continued to see the patient with longer or shorter intervals until the fatal termination of his disease, five months later.

The most striking feature in the subsequent history was the ups and downs of the patient. There were periods of steady improvement so great as to delude both friends and physicians with the hope of recovery, to be inevitably followed by periods in which the condition was worse. I will attempt, with the aid of some notes taken at some of my visits, to give a picture of his varying state, as well as of new symptoms as they arose.

March 21. Patient had been improving since the time of my first visit March 3; has had less headache, mind mostly clear, no difficulty in speech, and appetite better.

March 26. Since last date, headache much worse, more difficulty in collecting thoughts. Hitherto relief of headache was attempted with phenacetin, etc., which was never more than partially successful, and, in a few instances, morphia grs. ¼ hypodermatically. To-day, on account of the bitter complaints of the patient it was decided to try what a few days relief by means of morphia given rather freely (hypodermatically) would accomplish.

April 3. The morphia appears to have had a very bad effect. He clamors for it repeatedly during the day, and, if the injection is delayed at all, becomes violent. Otherwise he has become much worse. His stomach has become so bad that for some days he has refused food almost entirely. He has not permitted them to clean his mouth, which has become foul and offensive. His mind has become very much confused. He constantly uses the wrong words in speech—paraphasia—and manifests great irritability. For some days it has been observed that he is likely to drop objects, cigarettes, etc., held in his right hand. The rapid and great change in his condition, together with the almost absolute abstinence from food, gives a very critical aspect to his case. He is getting about two grains of morphia daily, and pupils are rather small and sluggish. With the idea that a large part of the present state was due to morphia he was on this date removed to the hospital and given a special nurse.

April 4. Only ¾ grains morphia in 24 hours. Patient calls attention to impairment of sensation and impaired use of right hand. An object placed in left hand was recognized at once; put in right hand (eyes closed), it fell out again, apparently unnoticed. Plantar reflex: left side flexion; right tendency to Babinski, and much less response than on the other side.

April 8. Mercury discontinued, because of a very sore mouth, perhaps altogether due to the long time neglect. Now the mouth is being properly attended to, for his mental condition, as well as his stomach and whole condition, is much better.

April 15. Headache has been better, so that ¼-⅜ grains morphia daily suffice; mind clear; no apparent difficulty in speech. Uses his right hand fairly well. Stereognosis fair or good. Plantar reflexes indefinite right and left—a sudden jerking away of the foot. Takes a fair amount of nourishment, chiefly fluids. His pulse for days has been 100 or a little above. He read some moderately fine print. His mouth is again normal. The injections of mercury were resumed and continued a month or more.

April 20. Has been getting worse since last note. More headache; receives ¾ grains morphia, or more, daily. Mind somewhat confused. Considerable paraphasia. Uses his right hand little, or none. All this time he has been in bed. It is observed that he moves the right leg less than the left. Pulse 72 to 84.

April 21. Would remain in the hospital no longer; went home.

May 2. No marked change since. Received from ¼ to ½ grain morphia daily. (It is always the custom to put off an injection as long as his patience will permit. The injections are given only by his physician, another cause for ofttimes delay.) Mind and speech as last noted. Right hand, grasp very feeble, movements very ataxic. Complete astereognosis and loss of tactile sensation. Painful impressions fairly felt, and localized. Plantar reflexes: left, flexion; right, Babinski. Right leg weaker than the left, no marked impairment of sensation in right foot. The act of showing the teeth reveals a slight right-sided facial paralysis. Sensation in face normal. Patient vomited twice; not cerebral vomiting, but due to a bad stomach.

May 12. The knee-jerk is more marked on the right side, and there is a suggestion of ankle clonus on the same side.

June 6. There has been a more or less steady improvement since the last note. The paralysis of arm, leg and face has disappeared, or about disappeared. All qualities of sensation in the hand appear to be normal. He had been in bed since ill, at times not able to stand, but now he is out of bed again and walks with a fair degree of steadiness, and on this date he took a drive of some miles to the oculist's office. All the tendon reflexes are normal. The plantar reflexes are again indefinite. He suffers less with headache than any time since ill, a large part of the time being quite comfortable if not quite free of pain. He feels for the first time as though he were getting well, and as though his mind were beginning to resume its former vigor, takes some interest in his business, and dictates some business letters. The only objective symptoms are those of the eye, hemianopsia, and difficulty of reading. In trying to read he often guesses wildly, just how far the latter is due to poor vision it is difficult to say. Dr. Holmes' examination on this date reveals still very marked choked disks in each eye, though the process appears to be receding, with convex lenses of 1.75 dioptries his vision is 20—30. With +3.5 he reads small type though only a few letters at a time.

July 1. The past three weeks has been worse again, more headache, mind less clear, arm and leg becoming paretic, though less markedly so than as noted May 2.

July 6. Only counts fingers at six feet. The left (the retained) half of the field of vision contracted to less than half its normal size.

July 13. Condition improved the past two weeks. Right leg and arm (including reflexes) proximately normal. Chief mental condition to be noted is obstinacy and poor judgment. Insisted on a number of occasions of going some distance to his dentist, without pressing need, and contrary to advice, and now determined to visit friends at Mt. Lookout, Chattanooga.

July 26. Went to Chattanooga July 14, and returned a few days later very much worse. While he walked fairly well when he left, he could not walk at all unsupported on his return.

On this date I find him quite somnolent, pulse 72, though Dr. S. says for several days it has been 60 or less. The right upper extremity appears to be completely paralyzed; when the sole of the paralyzed foot was pricked with pin it was jerked away—no farther test of power of leg possible in present mental state.

No ankle clonus, no tendon reflexes elicited in the arms. There is complete paralysis of left third nerve, pupil large, eye looking directly in front; the only possible movement is outwardly. Dr. S. first noticed this paralysis of third nerve a few days ago. Patient is about blind. Does not recognize any one by sight; but striking at the open eye causes him to wink.

August 4. Is absolutely blind. Paralysis of 3d nerve as before noted. Right arm flexed at all joints and rigid. Has been so a few days. Ankle rigid. Is delirious. Pulse for some days varied from 60 to 100. Breathing at times bad, with tendency to Cheyne-Stokes.

Died August 9, 3 a.m. The last few days the mental obtundity and bad breathing increased. He was comatose about half a day before death.

There are a few points not mentioned or not clearly brought out in the preceding history. Most of the time the patient had a bad stomach, had to be fed carefully, at times vomited, but in all this there was, at no time, what might be termed cerebral vomiting.

Very frequently in the last month or two he was found with a slightly elevated temperature, 100 or more.

The aphasic symptoms presented were paraphasia and, perhaps, alexia. To a slight extent the paraphasia was observed almost in the beginning. In his good periods it was absent or almost so. But in his bad periods it was very marked, and very annoying to the patient. It was manifested in using wrong names. If he called for a cigarette (during the whole period of his illness the craving for tobacco was always present) or a match, or a drink or the like, he was very likely to use the wrong word (book for cigarette, etc.), so that at times it was difficult or impossible to find out what he wanted. As to the alexia, in the beginning he read very well. He was not tested often on account of his headache, but at a later period, he would often guess wildly in reading, which could scarcely be accounted for by mere impaired vision, and not unlikely, indicated alexia.

I was not in the city at the time of the death of the patient, and did not see the autopsy. Unfortunately the specimen was spoiled somewhat in its removal. But a very large tumor was found occupying the larger part of the occipital lobe. A part of the apparent tumor was softened brain tissue. A microscopical examination showed the tumor to be a sarcoma.

At the time of my first examination the diagnosis of tumor of the occipital lobe was made. With such a diagnosis the question of operation naturally arose. Of course operation could not be advised before the efficacy of remedial measures, mercury and the iodides, had been tried; and the very rapid improvement, at different times, also, put the question aside. But apart from these factors, the case from the beginning did not seem a suitable one for operation. The sudden onset of hemianopsia indicated both a vascular and deep seated growth. The further history, great fluctuation of symptoms and appearance of motor and sensory symptoms seemed to verify the opinion that the tumor was vascular and deep seated, and to indicate, also, that it was extensive. For this reason though the question of operation was discussed with the family, an operation was not advised until after the examination of vision made July 6. It then became apparent that the patient was rapidly becoming blind, at a time, too, when his general condition was very good, so that one could not tell how long he might live. For that reason an operation (I had in mind either a large osteoplastic, with the object of seeking the tumor, or the mere removal of a piece of bone, for the purpose of relief) was strongly urged, but refused by the family. The autopsy shows that the radical operation could not have been successful.

AN UNUSUAL CASE OF CARCINOMA OF THE SPINE.[1]

By Philip Zenner, A.M., M.D.,

of Cincinnati.

The following case of carcinoma of the spine is reported because of the very long course of the disease, and to suggest a possible cause of this course.

Mrs. C., of Georgia, was forty-five years old when first seen by me, March 29, 1902. In December, 1897, Dr. Stark operated upon her for carcinoma of the (left) breast. In November, 1899, Dr. Stark examined her again and found a small mass in the left axilla, and edema of the left arm. He now removed both ovaries, influenced by Beatson's suggestion that oöphorectomy would arrest carcinoma of the breast. The edema of the arm subsided subsequent to this operation.

The history of pain—which continued to the end—begins with the fall of 1900, therefore nearly three years after the removal of the breast, and nearly one year after the removal of the ovaries. For a few months the pain was limited to the left leg, in the distribution of the anterior crural nerve, then it was felt in the other leg, also. Since then the legs have become weaker, the gait difficult and painful. The limbs have wasted considerably, the general health has suffered, and she has become very nervous.

At the time if my examination March 29, 1902, the patient was much emaciated, the lower extremities more wasted than the rest of the body, but the color was not bad—she did not present a cachectic appearance,—and her appetite was fair.

Her pains were variable in location—sometimes more in one leg, sometimes more in the other, or in the back—and intensity. Her manner of rising from a chair was something like that of the patient with pseudo-hypertrophic paralysis, that is she pressed with her hands against the thighs to help extend the knees. The gait was a slow shuffling one. The quadriceps group of muscles of each leg was paretic. Sensation was not appreciably impaired. The knee-jerks and Achilles tendon reflexes were normal.

There was tenderness over the spine, but also a somewhat diffused tenderness over the legs. The lower dorsal and lumbar spine was held quite rigidly. Flexing the spine produced a shooting pain on the inner side of the left thigh.

The pain and paralysis indicated intraspinal disease, while the impaired flexibility of the spine and pain in a distant part elicited by its movement pointed to disease of bone. The fact that this condition followed carcinoma of the breast led to the diagnosis cancer of the spine.

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[1] Read by title at the meeting of the American Neurological Association, September 15, 16 and 17, 1904.

The patient remained under my charge for about eight months, until November 20, 1902. At first there appeared to be considerable improvement in her condition, possibly due to the fact that, while prior to my seeing her she had been hobbling about the house, crawling up and down stairs with much difficulty, I permitted her to move about but little. The pain became less severe, the tenderness less pronounced, and there appeared to be more flexibility of the spine. But there was no permanency to this improvement; it was of but a few weeks' duration. The pain became again very severe, mostly in the left leg, less in the right leg, and little in the back. The condition remained variable. There would be periods of days when she would be better and worse.

July 19, 1902, an examination was made under an anesthetic, when it was observed that the left hip was much more prominent than the right, the trochanter being much enlarged.

The patient at this time was confined to her bed.

The next few months there was little to note in the general condition of the patient. Her general health remained much the same. Only it was found that she gradually required more anodynes. When I first saw her she was only taking small doses of codeine. Before she left us, she was getting morphia grs. ss and codeine grs. ss daily. Any considerable movement was very painful, and for that reason no thorough examination was again made, until the patient was about to leave for her home. There was at that time a large gibbus over the lower lumbar vertebra. The lower part of the spine was quite rigid. The left hip appeared to be dislocated, the upper part of the femur enlarged, the whole giving the impression of a dislocation due to a tumor. The greatest pain and tenderness were about the left hip. There was no manifest paralysis, for she could move every joint, though she did so very tenderly on account of the great pain engendered, so that the degree of strength could not be tested. There was no sensory paralysis. Patient was taken to her home in Georgia November 21, 1902.

Her husband wrote me March 15, 1903, stating that up to that time there had been no material change in her condition since she left Cincinnati. She died June 6, 1903.

Dr. Adams, of Madison, wrote that he saw the patient about April 15, that she was suffering with excruciating pains in the back and limbs, had lost the use of the lower extremities. She soon developed paralysis of the bladder and rectum, as well as large bedsores penetrating to the rectum and the spine. But there was no fever and the respiration, circulation and digestion were not markedly affected until toward the end. No autopsy was made.

The "paraplegia dolorosa" following carcinoma of the breast, makes the diagnosis in this case, cancer of the spine, a reasonably safe one. It is true the absence of an autopsy leaves room for doubt, but I shall, nevertheless, in what I am about to say, take that diagnosis for granted.

With this proviso, one clinical feature is, so far as I know, unique, that is the long duration of the spinal disease—from the fall of 1900 until June, 1903, nearly three years. I have seen a number of cases of cancer of the spine following disease of the breast, and in all of them the duration of the spinal disease, that is, counting from the time the pains began, was only a few months: and, so far as I can learn, this is the general experience.

The question arises in my mind, did the removal of the ovaries account for the slow course of the spinal disease?

In 1896 Beatson of Glasgow reported cases in which removal of the ovaries caused the disappearance for some time of cancer of the breast. This report gave rise to considerable discussion of the subject, a large part of which was unfavorable to such a view. But Boyd, in 1900, reported the results in 54 cases, in 19 of which there was more or less benefit. In some the cancer had not returned though several years had elapsed; in others the duration of the disease was more or less prolonged.

In the case reported in this paper Dr. Stark removed the ovaries because of the swelling in the axilla and edema of the arm, which subsequently disappeared.

In the first case reported by Beatson, there appears to have been a subsequent cancer of the spine, the duration of the latter about a year—an unusually long course, though much shorter than in my case.

I wish to raise the question, Was the long course in this case due to removal of the ovaries? Future observations in this direction may help to answer this question.

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

October 4, 1904.

The President, Dr. Pearce Bailey, in the Chair.

A Case of Unverricht's Type of Family Myoclonus, but Without Epilepsy—Presented by Dr. L. Pierce Clark.—The patient was a married woman, twenty-six years old, of Irish descent. There was a family history of myoclonus in the father, uncle and sister; of facial tic in a grandmother, of hysteria in the mother, and of paralysis agitans in a great-aunt. The patient was well until her seventh year, at which time the myoclonus began. It consisted of arhythmic, clonic spasm of the left sterno-cleido-mastoid muscle, and gradually spread into the entire left shoulder girdle, as well as the arm, trunk and thighs. Subsequently the right side became affected in a similar manner. The muscles most markedly affected were the sterno-cleido-mastoid, the trapezius, the deltoid, biceps, latissimus dorsi and glutei. The right side was most involved. Emotional excitement, embarrassment and anxiety increased the spasm. The gait was at times affected. The spasm was quieted by lying down. For the past eight years the spasm had been more or less continuous, with varying grades of paroxysmal intensity. It ceased during sleep. Speech was occasionally affected, and swallowing was at times difficult. Laryngeal sounds were occasionally emitted. The patient had never suffered from epilepsy nor from any attacks epileptoid in character.

Dr. B. Sachs asked how a case of this character could be distinguished from the *maladie des tic convulsifs,* to which the clinical picture bore a close resemblance? In Dr. Clark's case there did not seem to be anything more than a generalized convulsive tic, and without a distinct history of epilepsy he did not see how it differed from the affection described by French writers.

Dr. Clark, in closing, said the question raised by Dr. Sachs had given rise to considerable discussion between the French and German schools. The case resembled Unverricht's type of family myoclonus, in that the movements of the muscles were arhythmic, they were not purposeful, they were accompanied by paroxysms of intensity, but with no free periods. There was, however, no history of epilepsy, and some writers would no doubt classify the case as one of generalized tic.

A Case for Diagnosis—Presented by Dr. Sachs.—The patient was a man of thirty-five years, who first came under the speaker's observation in 1897. He was a machinist by occupation. He had never come in contact with metallic poisons, and gave no history of gonorrhea or syphilis. He first noticed that pulling the left half of his moustache caused him no pain, while pulling the opposite half gave rise to the usual sensation. Subsequently he had a carbuncle on the left side of the lip, which was opened without causing him any pain. Thorough and repeated examinations revealed nothing excepting a disturbed sensation in the distribution of the left trigeminal nerve. During the first year there was only a loss of pain and temperature sense in the greater part of the three branches of the nerve, with subsequent loss of sensation of the left cornea, and more recently, atrophy of the left masseter muscle. During his last examination of the patient, Dr. Sachs said, he noticed a slight involvement of the middle branch of the right trigeminus. There was no dissociation of sensation in any other part of the body, and no evidences of systemic or other diseases. The knee-jerks were normal.

Dr. Sachs said he had seen two cases similar to this one, in which the only symptom consisted of a dissociated sensation of the trigeminal nerve. One explanation he had to offer was that it was an unusual case of syringomyelia, in which the cavity formation was in the pons and not in the spinal cord. This he regarded as a rather venturesome diagnosis, because in such a case, in all probability, some of the other cranial nerves would have become involved after so many years. Another and more likely explanation was that the case was one of primary degeneration of the nerve, perhaps fibroid in character, such as had been described as occurring in nerves in other regions. Whether it would be proper to call it a neuritis or not he did not know.

Dr. George W. Jacoby said that one of the diseases to be considered in connection with the case shown by Dr. Sachs was beginning tabes. The speaker reported a case of undoubted tabes that had been under his observation during the past eighteen years, in which the first symptom was an analgesia in the distribution of the middle branch of the trigeminal; there was not a dissociation of sensation, simply a reduction, which gradually became complete, and this persisted for five or six years before any other symptoms of tabes manifested themselves. In that case, Dr. Jacoby said, anti-syphilitic remedies were given from the very onset of the analgesia, and for a long time, without exerting the slightest influence upon the subsequent development of the tabes.

Dr. William M. Leszynsky called attention to the decided atrophy of the muscles in the case shown by Dr. Sachs. The speaker said he saw no reason why the dissociated sensation should necessarily indicate the presence of syringomyelia. Dissociated sensation was occasionally seen in other conditions affecting the peripheral nerves.

Dr. Joseph Collins said that while cases of tabes like the one described by Dr. Jacoby were very uncommon, they were occasionally met with, and he had recently seen one come to autopsy after twelve years' observation. In those cases, however, there was no pronounced muscular atrophy, which was such a conspicuous feature in Dr. Sachs' case, constituting, practically, a facial hemiatrophy. The more or less uninterrupted quivering or flickering of the muscles, particularly the masseter, as well as the distribution and course of the affection, militated against a peripheral trigeminal lesion. Dr. Collins said he was inclined to regard the case as one of central syringomyelia.

Dr. L. Pierce Clark said that cases of progressive muscular atrophy had been reported which were similar to the case shown by Dr. Sachs. In those cases the skin itself was not involved to the extent that it was in facial hemiatrophy. The case might be one of progressive muscular atrophy, with sensory changes due to degeneration of the sensory root.

Dr. Sachs, in closing, said he did not think the case had any features in common with ordinary facial hemiatrophy. For a period of five years this patient only presented sensory symptoms. The apparent atrophy was due to wasting of the muscles supplied by the motor branch of the trigeminus. The quivering of the muscles, to which Dr. Collins had called attention, was more apparent than usual, and was probably due to temporary nervousness. The speaker said he had kept the possibility of early tabes in mind, but had found no evidences of that disease.

Dementia Præcox: Has the Recognition of Dementia Præcox Advanced our Conceptions of the Various Forms of Mental Derangement Occurring in the Adolescent Period? Is Not the Present Tendency to Give Too Grave a Prognosis in the Youthful Insanities? How Frequently Does Dementia Follow the Mental Derangements of Early Life?—Dr.

B. Sachs, in opening the discussion of this subject, emphasized the fact that some cases corresponded accurately to the types of dementia præcox described by Kraepelin and his followers. This was particularly true of the earlier forms of mental derangement occurring in members of families in which there was a very marked psychic taint, but even in these many years might pass before an appreciable dementia set in. The term dementia præcox, he thought, should be carefully restricted to such cases in which mental deterioration, at an early stage of the disease, was clearly recognizable, and should not be applied to those in which a dementia might set in in the far-distant future. The diagnosis of dementia præcox placed the stamp of an uncurable malady upon persons who might be sufficiently alert to be useful to themselves and to others for a long term of years, and in that sense did them distinct injustice. There seemed to be little gain in grouping widely differing cases under one heading. The older plan of clinical subdivision was more commendable, and the tendency to dementia should be insisted on only when there was reason to think that a deterioration was certain to develop at a relatively early period.

Dr. Adolf Meyer said that the question under discussion was a very broad one. Largely through the influence of Kraepelin's earlier writings, the speaker said he had come to regard katatonia and hebephrenia as degenerative conditions, and he was greatly surprised to see, in the fifth edition of Kraepelin's book, that the writer had apparently entirely changed his views on the subject, and had completely eliminated the degenerative features of those psychoses. Indeed, the very mention of degeneration to members of the Heidelberg school in 1896, when this great change took place, was looked upon as a sort of heresy. Instead of regarding these diseases of a degenerative origin, it was stated that they might befall anybody, just as general paralysis might befall anybody, and that they tended to end in dementia. Dr. Meyer said he had always been inclined to believe that Kraepelin's rather sudden change of views was due to controversial reasons rather than to an actual conviction. The chief advantage that had been gained by Kraepelin's writings was that it had induced psychiatrists to break away from the old tradition of classifying all forms of insanity into melancholia, mania and paranoia, thereby neglecting many well-marked and striking forms of mental disease which could be readily recognized. Undoubtedly Kraepelin went too far, a fact that he himself admitted when he said that the entire group was only classified provisionally, and would have to be still further subdivided. Kraepelin's description of dementia præcox was, on the whole, remarkably clear, and he was very painstaking in making the diagnosis. In a given case all the symptoms might not be present, yet the patient might show some evidences of deterioration. Such cases might be regarded as allied to dementia præcox if the symptoms pointed sufficiently in that direction.

Dr. William Hirsch said it seemed to him that this controversy concerning dementia præcox, as well as some other modern diseases, was largely a controversy about names. If it were not for the name dementia præcox, such a discussion would be practically unnecessary. The term did not originate with Kraepelin, but was used by Pick, of Prague, fully thirty years before, to describe a condition very similar to that to which it had been applied by Kraepelin. The clinical picture of dementia præcox is so broad that nearly every juvenile case of insanity would fit into one of the various categories, and the question arises whether there is really one important feature which all these cases have in common. Dr. Hirsch said they surely had nothing in common in symptomatology, because the clinical picture was too broad. Was the dementia a common feature of dementia præcox? Personally, the speaker said, his experience coincided with that of Dr. Sachs. He could not convince himself that all the juvenile psychoses, whether of the katatonic or other types, would terminate in dementia. There was one feature, however, that was common to all these cases of acute juvenile psychosis, namely, that they all occurred on a degenerative basis. We could always draw the conclusion that we had not to deal with a normal individual. These juvenile psychoses only occurred in individuals with inferior mental powers, and this fact should always be borne in mind in connection with the prognosis. Such a person might return to his relatively normal state—that is, to the condition he was in before the onset of the attack, which was never an absolutely normal condition. The fact was often lost sight of that these people never had a normal mind, but taking that for granted, it went without saying that any acute mental disease might intensify this mental inferiority, and that the patient might be worse off than he was before. Even if very decided dementia were present during the acute stage of the attack, it would not necessarily be a permanent condition: the patient might recover from this dementia and regain his relatively normal condition. The impaired mental condition of these patients, Dr. Hirsch said, did not necessarily imply a lack of intelligence, but might be characterized by hypochondriasis, by a lack of mental equilibrium or by morbid impulses.

Dr. Emmet C. Dent said that a recent analysis of the statistics at the Manhattan State Hospital for the past year showed that out of 911 patients admitted, 290, or about thirty-one percent., were cases of dementia præcox. The speaker said that after several years' study of Kraepelin's theory he was still inclined to believe that it was premature to discuss it. The group of cases included under the term dementia præcox was a large one, and in the course of time more appropriate names would doubtless be found under which the various types could be classified. A case of dementia præcox, when properly defined, was just as distinct a type as depressive insanity.

Dr. T. P. Prout said that five or six years ago he made some examinations of the blood in cases of so-called dementia præcox, and found that the results he obtained were very variable. In some of the typical cases, during the early stage, there was marked leucocytosis, while in others the number of leucocytes was decidedly diminished. A similar discrepancy was noticed in the differential count. It, therefore, seemed to him that different types of mental disease were embodied under this head.

Dr. Allen Ross Diefendorf, of Middletown, Conn., said that melancholia was recognized as a psychosis of involution, while dementia præcox was one of adolescence, and he could see no close relationship between the two. The symptoms of melancholia, as at present understood, were well defined, and it could be safely said that the condition was very rarely encountered during the period of adolescence. A review of about one-half the cases admitted to the Connecticut Hospital for the Insane during the past five years showed about 184 cases of dementia præcox. A subsequent review of those cases showed that twelve were mistaken for melancholia. Of these, seven were of the katatonic type, or were later recognized as katatonia, and five as hebephrenia. The speaker thought there was very little danger of confounding melancholia with dementia præcox.

Dr. Joseph Collins said he did not agree with Dr. Sachs that the modern psychiatrist showed a tendency to make the diagnosis of dementia præcox with alarming frequency, nor did he agree with Dr. Hirsch that there was anything typical in the clinical picture of this psychosis, or anything in common between the various types of the affection, as described by their originator. Furthermore, he did not think that Kraepelin maintained that position. In his study of the writer's work he could recall no ground for the statement that all these cases occurred on a degenerative basis. On the contrary, Kraepelin maintained that dementia præcox was a disease that occurred in individuals who were apparently sound, and that it was due to an auto-intoxication.

Dr. Edward D. Fisher said he was inclined to think that Kraepelin allowed a wide latitude in his differential diagnosis of dementia præcox, and that his cases were rather younger than those cited by Dr. Sachs. For example, take an apparently normal individual of fourteen, or sixteen, or eighteen years, who suddenly, with or without a psychosis, may show himself unfit for the work he is doing at the time, and is never able to take care of himself after that. We may have here a type of dementia which may never reach an extreme degree—that is, never go on to amentia. Such cases, Dr. Fisher thought, represented a large class to which Kraepelin referred in his writings.

Dr. Dent, in reply to a question, said that the cases of dementia præcox he had seen ranged in age from fourteen to twenty-five years. The average age was about twenty.

Dr. Sachs, in closing, said that the figures quoted by Dr. Dent showed that a large proportion of the patients at the Manhattan State Hospital were being admitted under the diagnosis of dementia præcox, and he questioned whether many of those cases had gone on to a condition of complete dementia. The only point he wished to make was to throw a little doubt upon the extremely grave prognosis that had been given in this class of psychoses in recent years under the teachings of Kraepelin.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

October 20, 1904.

The President, Dr. G. Alder Blumer, in the Chair.

A Case of Cerebral Tumor.—This was reported by Dr. Webber. The patient was seen about one year ago. She said she had been to the Massachusetts General Hospital six years previously. Her first illness was a convulsion just before going to the hospital. The left wrist and fingers were affected. Convulsions had been more or less frequent since, always of the same character. The left wrist, fingers and forearm were flexed; not always was there loss of consciousness. After she was seen by Dr. Webber she had no more attacks until near the close of life. At first there was no ankle clonus, no Babinski phenomenon; but later these became very marked.

At the autopsy no disease elsewhere than in the brain existed. The right anterior central convolution was much wider than the left. A section through the brain following this convolution showed the subcortical tumor, about one and one-half inches in diameter, situated in the motor area for the wrist, fingers and elbow.

The diagnosis was a tumor not in the cortex but in the motor tract. It was thought to be probably in the corona radiata, because there was more paralysis of the left arm and later of the leg, than is usually found in tumor pressing on or invading the cortex; there was less headache than usually attends tumor of the surface of the brain, and the attacks were less frequently generalized.

Les Crises de Psycholepsie, of which the following is a résumé of remarks made by Dr. Pierre Janet: For several years, in our teaching, Professor Raymond and I have given the name psycholepsy to a certain number of psychological disturbances which frequently occur in the course of neuropathic conditions, and which play an interesting part in the interpretation of their active deliria.

These disturbances are particularly clearly marked when they run a rapid course with a rather sudden onset; and they then constitute the psycholeptic crises. They are seen particularly in epilepsy and constitute one of those psychic equivalents of the seizures which have been described too often in a very confused way.

Observation 1. A young woman of twenty-two, with typical epileptic attacks since puberty; following an attack which was cut short, she suddenly goes into the condition now to be described: She sees and hears everything as well as usual; but although she feels as usual, she begins to have doubts of everything. She no longer knows whether what she sees really exists or not. Looking at her mother she says: "I should like it if that lady were my mother, but I can't bring myself to believe it. I can't seem to get it into my head that that lady is alive." She also has doubts about herself. She, too, has perhaps lost her personality, she isn't sure. She no longer believes anything she is told, and she cannot work nor concentrate her attention. This condition lasted four days; another time six days. It usually terminated in a typical epileptic fit.[1]

We may find many analogous examples in those patients whom I have described under the name of *psychastheniques,* and who show impulsive actions, obsessions, *delire de toucher,* and *folie du doute.*

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[1] A more exhaustive study of this class of cases may be found in my latest work, "Obsessions et Psychasthenie," vol. i. p. 506, and in the second volume published in collaboration with Professor Raymond, p. 56.

A patient described by Ball in 1880 said: "Suddenly I felt myself grow small and disappear; there was nothing left of me except my empty body with the same external form as before; since then my personality has entirely gone * * * * everything around me has become queerer and queerer, so that now I not only do not know what I am, but am unable to appreciate what people call the existence of things, reality."

I have myself made a large number of similar observations.

Very often *foile du doute, l'agoraphobia,* and the various obsessions begin suddenly with a confusion of this kind.

These crises may be short and followed by a more or less complete restoration of mental activity as is usually the case with epileptics; or they may be the point of departure for a long mental enfeeblement. If this is the case the feelings engendered by such a diminution of mentality play a very important part in the make up of the delirium.

This state may at first be only of a negative character, there being no clear cut disturbances either of the various sensations, or of motion, or even, strictly speaking, of the intelligence. At the beginning there may be no true delirium.

The positive features are at first the feelings which the subject experiences in regard to all his cerebral operations, feelings which bring him to say that everything about him is unfinished, incomplete, and without reality.

Then one may note the disappearance of the will and power of attention, and the subject becomes incapable of adapting himself to the situation in which he may be placed. In a word, we may say that the higher functions of the brain, which allow of adaptation to reality, as well as the feeling of realty itself, have disappeared; and that the lower functions alone are active. It is a lowering, a fall of the mind.

It would be difficult to discuss in this connection the psychological interpretations of these mental states which have been advanced. It will be sufficient to indicate their clinical importance.

Dr. J. J. Putnam said that he was reminded by Professor Janet's remarks of the sudden paralysis of motion which is occasionally seen in epilepsy without any antecedent convulsion, or more than a slight one. He said he could bear witness to the sudden onset of abnormal psychical states, and referred to an interesting case reported to him some years previously by Dr. Cowles.

Dr. Adolf Meyer thanked the Society for the honor bestowed on him, and expressed his gratification over being able to attend this meeting and to hear Professor Pierre Janet. The visit of this illustrious investigator of psychopathology has illustrated a point on which he insisted, viz.: the great meaning of actual contact, of a living reality being brought within our reach. This is bound to have a sound influence on our somewhat one-sided tendencies of following the aspects of mental disease which point in the direction of definite diseases, such as many are directly inclined to regard as definite disease processes, or pathological entities. Professor Janet had sketched a group of psychic facts which evidently occur in many types and combinations. He had referred to epilepsy, to neurasthenia (if that is not already included in psychasthenia), and in a similar way we might point to its occurrence in what we now call manic-depressive insanity, and in other "entities." At one time it stands alone and is designated accordingly; at others it can be viewed under the perspectives of other units of presentation, a special reaction type, for some reason adapted to the form of another type which we have learned to appreciate in appropriate cases. A patient at the Worcester Hospital had been admitted several times with distinct attacks of psychasthenia, with doubts and mysophobia; her father is there with the third attack of periodic depression; his mother had suffered from periodic depression. In view of the tendency of exclusive use of the concept of disease-process, Professor Janet's lesson is a most timely one. It stands for a freedom with, and even a need of, multiple perspectives in the rating of the disorders we meet. That he declines anatomical and physiological paraphrasing of the directly observable mental reactions is another good point. He thus holds us to the ground which demands the investigation, instead of distracting us from it into imaginative constructions. The account which Dr. Cowles has given is a splendid illustration. Had the interest been directed to physiological or anatomical hypotheses, the case would have been sufficient as first stated. Studied on its own psychological merits it has been investigated more deeply and found to present a wealth of facts rather leading away from what it had appeared to be at first sight. From a type of sudden agoraphobia it became a case with peculiar vascillations, with both distress and exultation, and relatively little of the phobia formula. Altogether, this working together of many interests helps along in what the human race sorely needs, greater faith in a multiplicity of perspectives and the abandoning of too set and exclusive formulas in nosology, wherever the facts are really too complex to fit into too simple a scheme.

Dr. Walton said that apart from the psychological interest in these phenomena it is of great practical importance that we learn, as far as possible, correctly to classify them, in order that the precursor, or the equivalent, of epilepsy may be distinguished from the less serious, though equally alarming, temporary derangement of the higher psychical function which indicates, perhaps, the obsessive temperament. Dr. Walton was consulted some years ago by a professional man of great acumen, who had experienced for a few minutes an utter loss of memory and affairs, even including the personality of his wife, his consciousness meantime being unimpaired, his speech unaffected, he himself being at the time acutely aware of his loss of mental grasp. He was reassured with confidence, and the event has proved the innocuousness of the experience. It would be no light matter to add the fear of epilepsy to the already sufficiently burdensome reflections of an idio-obsessive, because he had experienced such a temporary luxation of the associative mechanism.

Dr. Webber said that the relation of psycholepsy to ordinary epilepsy is interesting. Dr. Walton had mentioned the fear a patient had that attacks of *folie du doute* might change to epilepsy. As an instance of the reverse, which gives additional force to the theory that the two are closely related, Dr. Webber mentioned the case of a patient who many years ago was under his care with well marked epilepsy, having grand mal and petit mal. The attacks ceased, and he has had none since. Three or four years he came to Dr. Webber much disturbed by doubts as to whether he had done right, had told the truth; doubts as to his honesty, as to the effects upon others of what he had done. There was not the slightest foundation for such anxiety or doubts. He was satisfied temporarily; but he came again and again with the same or similar doubts. For more than a year Dr. Webber had not seen him. In a mild way he had *folie du doute*. Was that the equivalent of his former epilepsy, psychic instead of kinetic?

Dr. Knapp wished to thank Dr. Janet for the honor he had done the Society by consenting to speak to them on so interesting a subject. It had seemed as though a distinction might be made in these forms of psycholepsy between primary and secondary lowering of the mental plane. In a number of cases of obsession, or morbid fear, anxious states develop, and then, secondarily, as a result of the anxiety, there is a notable lowering of the mental plane. These conditions may fairly be distinguished from other forms where there is the sudden lowering of the mental plane as a primary state without the previous anxious state.

Periscope

MONATSSCHRIFT FUER PSYCHIATRIE UND NEUROLOGIE

(Vol. 14, 1903, No. 5, November.)

1. A Contribution to the Question of Spinal Centers of Certain Peripheral Nerves in the Dog. M. Lapinsky.
2. The Medulla Oblongata of the Nycticebus Javanicus. D. M. van Londen.
3. Contribution to the Knowledge of Progressive Facial Hemiatrophy. O. Fischer.
4. The So-called Galloping Paralysis, with Remarks Upon the Symptomatology and Pathological Anatomy of this Disease. H. Weber.
5. Subsequent Remarks Upon the Reflex Movement in Diplegia Spastica Infantilis, Described in Heft 4 of this Volume. H. Oppenheim.
6. The Meeting of the German Natural Historians and Physicians in Cassel, September, 1903, with a Section for Neurology and Psychiatry. Lilienstein.

1. *Spinal Centers in Dogs.*—Lapinsky injured various peripheral nerves in dogs, either by resection or ligation, occasionally ligating at the same time the arteries, and then at the end of a week or two killed the dogs and made serial sections of the spinal cords, which were stained by method. In this manner he was able to determine exactly the centers for the various peripheral nerves. The results are described at length and then tabulated, but the tabulation is so long that it is necessary to refer to the original paper. He also reaches certain general conclusions which are of interest. First, that certain nerve trunks arise from the various groups of cells which usually belong to different segments of the spinal cord. On the other hand, each group of cells supplies various peripheral nerves. As a result the spinal centers cannot be sharply separated one from another. Second, the group of muscles of one of the segments of the limbs obtains its fibers also from various groups of cells situated in various segments of the spinal cord. These groups of cells also supply other nerve plexuses. Therefore, it cannot be said that the centers of the plexuses have any individuality. Third, it is impossible to determine the position of the centers for the various nerve trunks, either histologically, physiologically, or even as a result of pathological and anatomical investigations. Consequently, the motor functions of the spinal cord cannot be localized, either according to spinal centers or to the centers of a peripheral nerve. Fourth, the contention of Brissaud, De Neeff, Nellis, van Gehuchten and other authors, that certain segments of the extremities are dominated by centers of nuclei which are isolated in the spinal cord, is difficult to prove. It must, of course, be remembered that Lapinsky's work applies only to the dog.

2. *Comparative Neurology.*—Van Londen gives a careful description, well illustrated, of the medulla oblongata of the *Nycticebus Javanicus.* It is not adapted to abstraction.

3. *Facial Hemiatrophy.*—Fischer reports an additional case of progressive facial hemiatrophy. At seven years of age the patient had had scarlet fever, and a year later a brown spot was noticed on the right side of the neck. This gradually enlarged, and atrophy of the right side of the face commenced. There was paralysis of the muscles of the face and the usual changes in the skin. The changes appeared to have commenced

at the embryonal points of closure in the neck and face. An analysis of forty carefully reported cases in which atrophic spots were present in the skin, showed that in thirty-three the spots were present at the embryonal points of closure, and in thirteen of these cases, also in other points. Fischer discusses the other causes, especially traumatism and the trophic nervous disturbances. He believes that we have two factors: a general and a local. The general, he agrees with Moebius, is probably a toxin, and the local either a congenital disturbance or an injury received later in life.

4. *Galloping Paralysis.*—Weber gives the following characteristics of galloping paralysis. The course is shorter—usually from one-half to one year. The death must be due to the disease and not to some complication. After a brief prodromal stage there is delirious excitement, confusion, disturbance of consciousness, and in a short time death. The physical signs consist of facial paresis, disturbance of speech, ataxia and changes in the reflexes. Pathologically there is found hyperemia of the blood vessels, round cell infiltration in their walls, and the appearance of neuroglia cells in the cortex. He reports a case occurring in a man thirty-four years of age, who had had syphilis. He was degenerate. His character had always been peculiar, and in June, 1902, he became gradually excited. In the latter part of July he had delusion of grandeur, rapidly grew worse, and in January, 1903, died. The microscopical examination of the brain showed a superficial leptomeningitis; no especial changes in the blood vessels, neuroglia or nerve cells, and there was a slight disappearance of some of the nerve fibers.

5. *Diplegia Spastica.*—Oppenheim discusses the reflex movements observed by him in two cases of spastic infantile diplegia, and states that he found them also in a case of post-epileptic coma occurring in an old woman.

(Vol. 14, 1903, No. 6, December.)

1. The Composition of the Posterior Columns of the Spinal Cord. K. Goldstein.
2. A Case of Acute Uncomplicated Paralysis of Touch. A. Knapp.
3. Contribution to the Clinical Knowledge of Periodic Psychoses. A. Pilcz.
4. Studies of Metabolic Changes Resulting from Cerebral Activity and Prolonged Wakefulness. Mainzer.
5. The So-called Galloping Paralysis, with Remarks Upon the Symptomatology and Pathological Anatomy of this Disease. H. Weber.
6. The Ninth Assembly of the Middle German Psychiatrists and Neurologists in Leipzig, October, 1903. H. Haenel.
7. The Seventy-fifth Meeting of the German Natural Historians and Physicians in Cassel, September, 1903, with a Section for Neurology and Psychiatry. Lilienstein.

1. *Posterior Columns.*—Goldstein has made a careful study of the tracts of so-called endogenous fibers in the spinal column, namely, the dorsal mediosacral tract of Obersteiner, the oval field of Flechsig, the tract of Hoche, the comma tract of Schultze, and the so-called ventral field. He had the following cases: One of compression of the corda equina without injury to the spinal cord, and one of tabes dorsalis. The microscopical changes are minutely described, and each of the five tracts mentioned subjected to special analysis. He then attempts to determine whether all these tracts are essentially the same, and whether they contain endogenous or exogenous fibers. It appears that there is a communication between the fibers in the sacrolumbar tract and in Hoche's and Schultze's tracts. Although it is impossible to answer the second question definitely, Goldstein is of the opinion that it is more likely that the tracts are made up of

exclusively exogenous fibers. The literature has been very carefully studied.

2. *Touch Paralysis.*—A man of forty-four years was bitten in the leg by a dog, which may have had hydrophobia. There was local suppuration. Seven years later he suddenly lost the power of sensation in the left hand. This returned in two weeks. Later he had an attack of clonic spasm of the left side of the face, lasting for about three hours. Still later he had a feeling of numbness in the left hand. His general health was excellent. When examined it was found that he had complete loss of the stereognostic sense in the left hand. It seems likely that the cause was a small hemorrhage into the parietal lobe.

3. *Periodic Psychoses.*—Pilcz reports a series of cases of periodic psychoses. One, a man of thirty years, with neuropathic heredity, had attacks coming on acutely, terminating by lysis, and always characterized by a preliminary loss of weight with increase towards the end of the attacks. The attacks were also chiefly characterized by hallucinations of sound, and from time to time paroxysms of rage, with cyanosis. In the second case, a Jewess of fifty years, with neuropathic heredity, the disease commenced at the age of thirty-eight acutely, with manifestations of melancholia. Later there was an attack of mania and then lytical recovery. The duration of the melancholic period was more than four years, and the maniacal nearly as long. The second period followed almost immediately after the first, and the patient did not recover until June, 1903. During the period of recovery there was considerable increase in the body weight. Pilcz gives the subsequent history of some cases that he had previously reported. In one of these recovery seemed to be due to a suppurative periostitis of the jaw. Altogether he has collected seventeen cases in which an intercurrent disease appeared to produce recovery.

4. *Metabolism in Disease.*—Mainzer has performed some very elaborate experiments in metabolism in order to determine the effect first, of intellectual work, and second, of prolonged wakefulness. The results were somewhat disappointing. The relation of the phosphoric acid to nitrogen showed no constant variation. Prolonged wakefulness did not increase the amount of the imperfectly oxydized nitrogen in the urine, in fact, distinctly the opposite occurred. At present a sufficient number of experiments have not been made to enable us to determine exactly what changes occur.

(Vol. 15, 1904, No. 2, February.)

1. Histological Investigations Upon the Nerve Endings in the Tendons and in the Perimysium of Rats and Guinea Pigs. G. Cabibbe.
2. Experimental and Pathologico-anatomical Investigations Upon the Course of Certain Nerve Tracts in the Central Nervous System. G. Mingazzini.
3. The Neuro-pathological Valuation of Nocturnal Enuresis and Similar Disturbances. H. Pfister.
4. Certain Defects and Difficulties in the Classification of Mental Diseases. Th. Ziehen.
5. The Origin and Course of the Oculo-motor Nerve in the Mid-brain. St. Bernheimer.

1. *Nerve Endings.*—Ruffini's method consists in placing fragments of non-parenchymatous tissue first in a solution of formic acid and then in a solution of gold chloride. After the impregnation is complete the tissues are teased and mounted on cover glasses. Two types of sensory bodies were found, the Pacini and the Golgi bodies, or spindles. They showed an extraordinary diversity in structure and arrangement. Occasionally the different bodies appeared to be in contact, although Cabibbe expressly refrains from stating that they are united.

2. *Nerve Tracts and Endings.*—Mingazzini describes the histological changes in five additional brains. In the first, a monkey, the left half of the cerebellum, a portion of the vermiform process, and part of the left half of the medulla oblongata were removed three years before the animal was killed. For the details of the histological changes of this and the following cases the reader must be referred to the original. The second case was a man who had observed one morning that his mouth was drawn to the right. Later his mind weakened, and he had periods of maniacal excitement. There was some paresis of the left side, and some disturbance of touch and pain in the same region. Romberg's phenomenon was present; there was slight dysarthria and paralysis of the sphincters. The patient died of chronic external pachymeningitis, and a hemorrhagic cyst was found in the right thalamus. The microscopical examination is given minutely. The third case, a boy of seventeen, had commenced to have epileptic attacks at the age of five or six years. In walking the patient fell to the right and forward. There was almost complete anesthesia on the right side. The epileptic attacks were frequent. A tumor was found springing from the surface of the right caudate nucleus and involving the right optic thalamus. The posterior portion of the third ventricle was obliterated. This tumor consisted of neuroglia. The changes in the various tracts are carefully described. The fourth case was a female infant suffering from microcephalus, that died at the age of two months. The weight of the brain was 105 gm., the circumference of the head 270 millimeters. The diminution in size chiefly concerned the cerebral hemispheres, of which the right was smaller than the left. The pyramids were entirely absent. On the left side the island of Reil was exposed, and showed no convolutions; the frontal lobes were comparatively strongly developed. The occipital lobes were entirely deficient. The fifth case, a man of forty-one, was born with paralysis of the right side. He was an imbecile from birth, was usually apathetic, occasionally excited, speech was confined to a few simple words expressing primitive ideas; he had never learned to write. There was paresis of the left side of the face. A cyst about the size of a pigeon's egg was found in the left frontal lobe. There was atrophy of many of the structures on the left side, and the anterior horn of the left ventricle was dilated. The pyramidal tract in all parts rising from the left side of the brain was atrophic. A small area of softening was found at the base of the left posterior corpus quadrigeminum. The paper is still unfinished.

3. *Nocturnal Enuresis.*—Pfister reports twenty cases of nocturnal enuresis. He divides them into two groups: first, characterized by the fact that control of the bladder was not acquired as the child advanced in age; second, in which enuresis did not develop until comparatively late (after the fifth year), and then was not common. In this latter group the enuresis can usually be regarded as an accompanying sign of epileptic discharges, rarely of hysterical attacks. Essential enuresis occurs most frequently at night, occasionally in the daytime, and is often aided by various emotional disturbances, such as laughing, or by convulsive movements, such as sneezing or coughing. It is not a symptom of any particular disease, but is really a stigma of disturbances in development, either in the form of a slow completion of some of the tracts, or of a disposition to some nervous or psychical disease. It often occurs as a familial disturbance.

4. *Classification of Mental Disease.*—Ziehen calls attention to certain requirements in a study scheme for the classification of mental disease. He discusses the difficulties of an etiological classification. There is great difficulty, too, in classifying properly the emotional forms of psychoses and transitional forms. More attention should be paid to the individual variations.

5. *Oculo-motor Nerve.* St. Bernheimer writes a controversial article.

(Vol. 15, 1904, No. 3, March.)

1. Hysterical States of Confusion and Talking at Random. J. Vorster.
2. The Behavior of the Reflexes in Brain Tumors. A. Reh.
3. A Case of Speech Disturbance. W. Alter.
4. Porencephaly and Cerebral Infantile Paralysis: A Contribution to the Psychopathological Nomenclature. Sommer.
5. Experimental and Pathologico-anatomical Investigations Upon the Course of Certain Nerve Tracts in the Central Nervous System. G. Mingazzini.

1. *Hysterical States.*—Vorster reports five cases of hysterical confusional states, all showing marked neuropathic heredity. Four were men under twenty-five, and the fifth was a woman of thirty-four. For the symptoms in the individual cases reference must be made to the original article. The author briefly discusses the literature.

2. *Reflexes in Brain Tumors.*—Reh has tabulated 100 cases of brain tumor with reference to the reflexes, the clinical symptoms and the pathological findings. He found that in thirty-four per cent. the patellar reflexes were diminished; in thirty-six per cent. they were increased, and in the remaining cases were normal. In a few of the cases the diminution was unilateral, and usually on the same side as the tumor. The position of the tumor did not appear to be of great influence. Increase in the brain pressure sometimes caused diminution of the reflexes. Degeneration of the posterior columns of the cord was present in twenty-two of the thirty-two cases examined. In the majority of these cases tumors were found in the cerebrum, and choked disc was present. In some cases the reflexes were diminished, but in only a slightly less number they were increased. The literature is very complete.

3. *Speech Disturbance.*—The patient had had an apoplectic attack followed by brachial monoplegia, hemianopsia and aphasia. The latter was of the type of dysarthria. From time to time he had attacks in which he would remain quiet for some time, then commence to articulate in a loud voice various combinations of the R with the vowels, the rhythm of his speech resembling that of ordinary speech composed of words and sentences. When he attempted to repeat words he used the same combinations, but there was a distinct effort at imitation. Later he was able to add a K to his combinations. The attack lasted about ten days, when the patient returned to his previous dysarthric condition. Several subsequent attacks occurred in which the speech was limited to a few consonants and the vowels. Alter regards the lesion as probably cortical in nature. The curious attacks are probably glosso-psychical in nature. He offers some theoretical considerations regarding the inter-relation of the higher psychical and the speech centers.

4. *Porencephaly.*—Sommer contributes an article upon the significance of the word "porencephaly." He regards it as indicating those disturbances of the brain associated with idiocy and epilepsy, and characterized by cavity formation in the brain, that is, a defect of the brain substance.

5. *Nerve Tracts*—Mingazzini continues his careful description of the central nervous system. The paper is still unfinished.

(Vol. 15, 1904, No. 4, April.)

1. Retrograde Amnesia after an Attempt at Strangulation, and After Injury to the Head. E. Hess.
2. A Case of Touch Paralysis and Jacksonian Epilepsy, and the Favorable Results of the Removal of Adenoid Vegetations. A. Knapp.

3. Experimental and Pathologico-anatomical Investigations Upon the Course of Certain Tracts of the Central Nervous System. G. MINGAZZINI.
4. A Case of Psychical Disturbance of the Motor Functions with the Predominant Involvement of Speech. W. FÖRSTERLING.
5. Neurasthenic Melancholia. M. FRIEDMANN.
6. The Fatigue Curve of Healthy Persons and in Patients Suffering from Neuroses and Psychoses. BREUKINK.

1. *Retrograde Amnesia.*—Hess discusses the subject of retrograde amnesia after attempts at strangulation, and reports three cases. The first, a man of thirty-six years, a chronic drunkard with neuropathic heredity, attempted to hang himself and was rescued, experiencing no bad effects. At a second attempt he was entirely unconscious when cut down and had no recollection of the attempt. His mental state resembled closely that of dementia præcox. A second case, a man of twenty-two years, injured his head while riding a bicycle. When he had recovered consciousness he had forgotten all the events of the day on which the accident occurred. Four months later his memory had returned completely. It is possible, however, that this return of memory was merely simulated, and that his knowledge of the events was derived from conversation with his family and companions. The third case, a woman of forty-two years, was injured in a railroad accident. There was a severe wound on the head, and more or less general shock. There was complete loss of recollection of all that had happened for some time before the accident. In three or four weeks her memory had returned, but she then lost the recollection of having had the amnesia. Hess is if the opinion that the return of memory in this case also was merely apparent, and that actually she had learned of all the events from conversation with others. The loss of memory of the amnesic period was probably due to insufficient introspective capacity. He also suggests a rather original explanation of some of these cases of retrograde amnesia. In the majority of instances the patients have been taking more or less severe exertion, or have been drinking—sometimes both, and he had observed that under these circumstances persons become drowsy and perception is diminished. This is particularly apt to occur during bicycle riding or marching. Of course, there is no recollection of what has been done during these periods of sleep with action. Retrograde amnesia is probably fairly common, but often the defect in memory is corrected before the physician sees the patient, in the manner in which it was apparently corrected in two of the reported cases.

2. *Touch Paralysis.*—A boy of seven had an epileptic attack followed by weakness and numbness of the left side. There was complete astereognosis of the left hand without loss of touch sense, and with partial loss of position sense. Adenoid vegetations were found in nose and removed. The symptoms immediately improved, but the epileptic attacks continued. The stereognostic sense, however, was greatly improved. About four months later the epileptic attacks had practically disappeared, and there was only a slight increase in the reflexes on the left side. Knapp supposes that there was a cortical lesion insufficient of itself to produce the epilepsy, but in the presence of adenoid vegetations, which interfered with respiration, the epileptic attacks could occur.

3. *Nerve Tracts.*—Mingazzini discusses some of the results of his investigations regarding the course of the fibers in the central nervous system. It is impossible properly to give these results in abstract.

4. *Psychical Disturbance and Speech.*—Försterling reports the case of a woman, thirty-seven years of age, with neuropathic heredity, who became restless, irritable and had attacks in which she screamed at regular intervals for considerable periods of time. There was a continuous disposition to speak. There was also an epileptic attack.

5. *Neurasthenic Melancholia.*—This syndrome is usually characterized by the simplicity of its symptoms. Friedmann discusses particularly the question whether there is a sharp differentiation between the neurasthenic melancholia and the true psychosis, and whether the distinction between the two conditions can be more exactly made. The paper is still unfinished.

6. *Fatigue Curve Studies.*—Breukink has employed the ergograph of Kraepelin for the purpose of determining the fatigue curves in various conditions. In hysteria there is often sudden interruption in the movements. In neurasthenia there is occasionally a concavity of the upper points of the line. In chorea the contractions are irregular, and in various forms of hemiparesis the movements are less frequent. In progressive muscular dystrophy the frequency of movements was nearly normal, but their amplitude decreased rapidly.

(Vol. 15, 1904, No. 5, May)

1. Contribution to the Knowledge of So-called Original Paranoia. A. SCHOTT.
2. Experimental and Pathologico-anatomical Investigations Upon the Course of Certain Tracts of the Central Nervous System. G. MINGAZZINI.
3. Neurasthenic Melancholia. M. FRIEDMANN.
4. The Cytodiagnosis of Tabes Dorsalis. H. S. FRENKEL.

1. *Original Paranoia.*—After discussing the literature, Schott reports the case of a man, forty-nine years of age, with neuropathic heredity, whose character had always been peculiar. At the age of twenty-four it was observed that his mind was affected. There were hallucinations and delusions of persecution and grandeur. The course of the case was irregular, and in the periods of intermission all recollection of the periods of hallucinations was lost. Many symptoms resemble those of dementia præcox, but the long duration of the case would render such a diagnosis unlikely. Schott prefers a diagnosis of original paranoia, and thus the case indicates the significance of the deceptions of memory in these conditions.

2. *Nerve Tracts.*—Mingazzini concludes his long and valuable article upon the minute anatomy of the central nervous system.

3. *Neurasthenic Melancholia.*—Friedmann concludes his article upon neurasthenic melancholia. He reports a number of cases, and concludes that in about two per cent. of cases of neurasthenia the emotional depression reaches a degree which renders them peculiar. He recognizes different groups. A first, in which there are delusions, from which he reports at length a number of cases; a second, with nervous apathy and stupidity, and a third, with severe conditions of irritability and anxiety.

4. *Cytodiagnosis in Tabes.*—Widal's method of studying the cerebrospinal liquid is to allow 3 or 4 ccm. to flow from the cannula introduced into the spinal canal, into a centrifugating glass. The centrifuge is then revolved at the rate of 2,500 to 3,000 times a minute for ten minutes. The fluid is then poured off, the glass held perpendicular, and the thick drop which adheres to the end of the tube drawn off into a small glass tube by capillarity. This is then spread upon a cover-glass, washed with absolute alcohol and ether until all the crystals are removed, and then stained with Unna's polychrome methylene blue or hematin and eosin. The characteristic of tabes in this fluid is the great excess of lymphocytes. In twenty-three cases Frenkel found this to be the case. For making the lumbar puncture Frenkel prefers a trocar and cannula, which is thrust in between the fourth and fifth lumbar vertebræ. If the point of the trocar strikes bone he prefers to withdraw the instrument completely. If blood flows the first few centimeters may be drawn away, and the fluid is then usually clear. In-

fection is not likely, even if the instruments are not thoroughly sterile. The same results are found in general paralysis.

J. Sailer (Philadelphia.)

BRAIN

(Vol. 26, 1903, No. 4, Winter.)

1. Case of Progressive Muscular Atrophy of Spinal Origin in a Girl Aged Eleven Years: with Autopsy. Herbert, Morley, Fletcher and Frederick E. Batten.
2. Beri-Beri in Monkeys. Hamilton Wright and M. D. McGill.
3. Associated Movements in Hemiplegia: Their Origin and Physiological Significance. H. Campbell Thompson and F. R. C. P. Lond.
4. Experimental Research on the Course of the Optic Fibers. George Dean and C. H. Usher.
5. Post-diphtheretic Chronic Bulbar Paralysis and Its Distinction from Myasthenia. Wilfred Harris.
6. Degeneration Resulting from Lesions of Posterior Nerve Roots and from Transverse Lesions of the Spinal Cord in Man. A Study of Twenty Cases. James Collier and E. Farquhar Buzzard.

1. *Progressive Muscular Atrophy.*—The authors contribute the history and pathological findings of a case of progressive muscular atrophy of spinal origin occurring in a child. M. L., eleven years. Twelve months weakness of hands; seven months unable to button clothes; four months weakness of legs; two months adenoids removed, and much worse since that operation; fourteen days pain in back and increased difficulty in swallowing: intelligent, deaf, some atrophy of tongue, palate moves well on phonation, weakness of neck, respiration thoracic, upper intercostals only in use; claw hand, wrist drop, weakness of upper arm and shoulder muscles; can walk but only feebly, knee-jerks brisk, plantars flexor, sensation natural to all forms; electrical reaction R. D. in some muscles, in others only diminution of irritability. Sudden death from respiratory failure six months later.

The pathology of the condition was extremely intricate. By the Marchi method degeneration was discovered commencing about the twelfth thoracic segment, increasing with the eighth and seventh, and reaching its maximum intensity at the sixth, the gray matter being hard to distinguish from the surrounding white matter. The fibers occupied the region of the cord all round the gray matter of the anterior horns and somewhat the antero-lateral tracts. Other studies by the Nissl and Weigert methods were made. The authors summarize their findings as follows: (1) There was extensive destruction and atrophy of the cells of the anterior horn from the upper cervical to the lower thoracic segments, this change being most marked at the level of the sixth thoracic segment. The cells of Clarke's column were also affected. (2) There was extensive degeneration of the whole of the ventral region of the cord in the thoracic segments, the lesion being most marked at the level of the sixth thoracic segment, and diminishing in extent both toward the medulla and caudally. There was extensive degeneration in the direct cerebellar tracts, both dorsal and ventral, beginning in the lower thoracic segments and extending up to their respective terminations in the cerebellum. The degeneration affected both gray and white matter alike in that region of the cord which was most affected, viz.: the thoracic segments. (3) There was a marked increase of the connective tissue in the ventral portion of the cord in the thoracic region (4) The walls of the vessels showed no change; there was engorgement of vessels with extravasation of blood into the perivascular spaces. (5) The cord showed both recent and old degeneration.

2. *Beri-Beri in Monkeys.*—The authors conclude from their studies that beri-beri is present in monkeys as well as in man, and summarize as

follows: The onset, early and developed symptoms, the underlying lesion in the stomach, duodenum and nervous system, the condition under which the disease was contracted, are so similar to what has been found in man that there can be no hesitation in concluding that all these monkeys contracted beri-beri. Case 3 alone would not have warranted such a conclusion, not because the animal had become septic, but because it had become so grossly septic. It would not be urged that beri-beri in man was not beri-beri, because a few days before sepsis had occurred as the result of infection through bed or other sores, or because of death being due to some intercurrent disease, such as phthisis or dysentery. No doubt sepsis and intercurrent diseases may modify to some unrecognizable extent the characteristic lesion of beri-beri in the peripheral and central nervous system, but that is all. On the other hand, there is no doubt whatever that the lesion of the nervous system in beri-beri induces disease in the lungs and other parts of the body, *e.g.*, phthisis, fatty degeneration of the heart, liver, etc. The fact, therefore, that Case 27, already published, and Case 3, included here, were septic during the few days before they were killed, does not militate against the conclusions, for the characteristic symptoms of beri-beri preceded the sepsis by weeks. Cases 27, 3 and 94 should be classified as acute, and 125 as sub-acute beri-beri, that would have recovered or have ended as residual beri-beri paralysis, had the animals not been killed during or shortly after the active stage of infection had ceased and the organism and its toxin eliminated. But Case 86 should be classified as acute pernicious beri-beri, in which the causal organism developed rapidly, producing so virulent a toxin that it acted widely and severely on the vital and ordinary afferent neurones, and so rendered the animal moribund in a few hours. Had it been given a chance, the authors do not think this animal would have recovered. The value of this observation lies in the fact that it conclusively proves: (a) That beri-beri is an acute infectious disease. (b) That the organism—as yet unknown—exists in close, sunless foci, *e. g.*, the cells of a jail in this instance. (c) That no food, as food, either qualitatively or quantitatively, is a factor in the production of the disease. (d) That the organism is not an organism that affects or develops on food commonly used by man. (e) That the organism is probably ingested with food accidentally contaminated by it. (f) That it multiplies in the stomach and upper part of the small gut, causing a local congestion or inflammation, and elaborates a toxin which, being absorbed, acts with varying force bilaterally and symmetrically on certain vital and ordinary neurones, to give rise to the collection of symptomes known as beri-beri. (g) That the incubation period of beri-beri is short. (h) That rice, either as a diet or as the habitat of a living specific organism, cannot for a moment be seriously considered. These conclusions were arrived at after a careful experimental study of the disease in man, and are confirmed by this observation on monkeys. It but remains to detect the organism. The authors are convinced that no good but only confusion will result if bacteriologists and other students of the subject do not firmly grasp the distinction made between beri-beri, the acute infectious disease, and its residual paralysis.

3. *Associated Movements in Hemiplegia.*—Thomson seeks to show that the involuntary associated movements seen in hemiplegia have a definite history in evolution, and were once useful for ordinary purposes of life. He goes back to the fishes and lower animals to show the origin of many of these movements, particularly demonstrating that the evidence goes to show that respiratory movements in animals were originally closely associated with movements of the limbs, and it seems likely that it is this association that accounts for the presence of commissural fibers between the nuclei which preside over the two movements. As the respiration becomes more thoracic in character its association with the legs would become weakened, and then, later on, the movement of the arms would gradually

become disassociated as they became educated to perform more special movements, for the inhibition required for these finer movements would effectually prevent any movement taking place with respiration, which, if allowed, would obviously destroy the utility of the arms for other purposes. At the same time the increasing automatic action of the respiratory movements would also tend to make ordinary respiration independent of any support from the limbs except in cases of difficulty, and thus the movements of respiration and of the limbs, at first intimately associated, would gradually drift apart until finally, with the development of inhibition on the one hand, and with the disuse of the commissural paths on the other, the association would practically disappear, but as soon as the inhibitory impulses are taken off, the limb again tends to resume its old relations, the unused paths become reopened and the old associations are revived. In health these paths have still, in this instance, been used a little during yawning, so that their obliteration has never been quite complete. Yawning is a peculiar respiratory act in many ways, and is certainly more automatic than ordinary breathing. According to Sir Michael Foster the stimulus in yawning probably acts upon the respiratory center itself; possibly the centers in the cord also take a share in it, but in any case yawning may be looked upon as a lower type of the respiratory process than ordinary respiration, and, therefore, the one with which the movement of the arms would be likely to be longest associated.

4. *Optic Fibers.*—The present communication gives the results of a number of experiments on monkeys with degeneration in the optic nerve and in the chiasm resulting from lesions of the retina or division of the nerve. The results obtained are extremely complex and do not permit of summary in this place.

5. *Diphtheritic Paralysis and Myasthenia.*—Chronic bulbar paralysis, occurring as a sequel to diphtheria, has been found by the author in only four instances. Nevertheless, he believes that very occasionally permanent palsy may result following diphtheritic paralysis. That when such permanent paralysis remains it is usually of muscles supplied by one or more of the bulbar nuclei. That a distinct type of bulbar paralysis may ensue, closely resembling in its distribution that which is familiar in myasthenia, or asthenic bulbar palsy, but which is to be distinguished from the latter by the non-variability of the symptoms, the absence of the myasthenic reaction, the absence of ptosis, or weakness of the jaw muscles or of the neck or limbs. It is further distinguished by the absence of the attacks of dyspnea, so characteristic of many cases of myasthenia, and by the presence of muscular atrophy and the reaction of degeneration.

6 *Degeneration from Lesions of the Spinal Cord.*—The authors have examined in this paper two cases of isolated lesion of the posterior roots in the cervical and lumbo-sacral regions, twelve cases of transverse lesion of the spinal cord at various levels. The paper is extremely elaborate and bountifully illustrated and should be considered in the original.

(Vol. 27, 1904, Spring.)

1. Hysteria and Neurasthenia. SEYMOUR J. SHARKEY.
2. A Case of "Subacute Combined Sclerosis," with Profound Anemia. JAMES TAYLOR.
3. The Effect of Total Transverse Lesion of the Spinal Cord in Man. JAMES COLLIER.
4. On the Primary Staining of the Rat's Brain by Methylene Blue. JOHN TURNER.
5. Note on a Case of Defective Development of the Lateral Cerebellar Lobes in a Dog. THOMAS LEWIS.
6. Unilateral Congenital Lesion of Medulla and Spinal Cord; Death from Pontine Hemorrhage. PURVES STEWART.

7. Binocular and Stereoscopic Vision in Man and Other Vertebrates, with Its Relation to the Decussation of the Optic Nerves, the Ocular Movements, and the Pupil Light Reflex. Wilfred Harris.

1. *Hysteria and Neurasthenia.*—In a presidential address delivered before the Neurological Society of the United Kingdom, Dr. Sharkey spoke of these two conditions. He spoke of (*a*) explosiveness, (*b*) want of control, (*c*) proneness to exhaustion, as characteristic of brain activity in hysteria, and then went over considerable old ground in delineating some of the clinical signs. If the disease, as he says, is to be looked upon as a congenital condition, one canot hope to cure it. Rest in bed, isolation of the patient, massage and tonics are all he can suggest in the way of treatment. In speaking on neurasthenia he believes that it is not impossible that a real degeneration of nerve cells takes place, and therefore the treatment requires considerable time.

2. *Subacute Combined Sclerosis.*—The history and pathological findings of a case of this disorder, occurring in a severe anemia, and similar in many respects to cases already spoken of.

3. *Transverse Lesion of the Spinal Cord in Man.*—Collier presents a paper drawn from the records of fifteen cases of transverse lesion. He defines transverse lesion as occurring when (*a*) the spinal cord is actually severed, (*b*) serial longitudinal sections, stained by appropriate methods, show no myelinated fibers, (c) transverse sections show whole areas to be necrotic. He remarks, with truth, that the size of the spinal cord in the region of the lesion is no criterion for the extent of the injury. Clinically, a physiological total transverse lesion may be considered as existing when there is absolute loss of sensibility, complete flaccid paralysis and loss of the deep reflexes. Such complete physiological abrogation of the functions may exist without the actual death of the elements involved. These elements may recover more or less completely. When there is complete degeneration of all elements of the transverse area of the spinal cord, there can be little doubt, he says, that sufficient continuity for the maintenance of the spastic state and for the return of the knee-jerks may be re-established by the regeneration of the conducting paths. With gradual lesions the order of appearance is practically as follows: Motor paralysis and spasticity, equilibratory anesthesia, sphincter paralysis, therm-anesthesia and analgesia, tactile anesthesia, flaccidity with loss of the deep reflexes, progressive muscular wasting, progressive lowering of faradic excitability, progressive loss of sphincter tone. The motor paralysis and spasticity develop slowly and with even steps. Sphincter paralysis is variable, sometimes appearing long before any skin sensory loss is apparent, sometimes long delayed until skin sensory loss is marked. Loss of skin sensibility from increasing lesion, when once apparent, progresses much more rapidly than does the motor paralysis, and it would appear that sensibility to pain and temperature is always impaired earlier and to a greater extent than is sensibility to touch. The change from the spastic state to the flaccid state takes place rapidly in from twenty-four hours to a week, according to the acuity of the lesion, and is accompanied, or shortly followed, by loss of the deep reflexes. It never occurs till the sensory loss is complete over the lower extremities. Muscular wasting, lowering of faradic excitability and loss of sphincter tone commence with the onset of the flaccid state, and are progressive as long as the physiological transverse lesion remains total. When there is recovery from the total physiological transverse lesion the first sign of such recovery is the return of the knee-jerk and a gradual change from the flaccid to the spastic state. Sensibility returns in the order, touch, pain temperature; and the lowest sacral segments usually become sentient before the others. The first sign of such return is often that the patient becomes conscious of the act of micturition, or of catheterization, or of the distention of the bladder during the routine lavage. In one case sensibility returned first in the feet.

Voluntary power returns in the flexors earlier than in the extensors, flexion of the toes being almost invariably the earliest voluntary movement possible. In cases where recovery is complete the plantar reflex does not resume the flexor type until some time after the patient has completely recovered the use of the lower limbs, the extensor response being invariably the last of all spastic signs to disappear. He then discusses in detail the condition of the distribution of the sensory loss; the evidence derived from the muscular condition, which from clinical evidence, he says, strongly supports the argument that muscular wasting, lowering of faradic excitability, and loss of deep reflexes under consideration, are the result of isolation of the lumbo-sacral region of the cord from the higher centers above. The deep reflexes are abolished below the lesion. Their partial return is evidence that the lesion has not remained total. Skin reflexes appear to be invariably abolished with the possible exception of the plantar reflex. The visceral reflexes in his series were variable, abdominal distention met with in a few cases, and edema and trophic changes were constant.

4. *Staining of Rat's Brain.*—The author gives a series of results obtained by various methods, using methylene blue as an intravitam stain. As the paper is very much detailed it should be consulted in the original.

5. *Defective Cerebellar Development.*—The author describes a typical case of circus movement in a dog in which it was shown that the abnormality was associated with destruction in the lateral lobes (and so with the pontine nuclei) alone. This case, in the author's opinion, lends weight to the view that the cerebellum may virually be regarded as a twofold organ, consisting of one part, the vermis, essentially spino-cerebellar, and another part, the lateral lobes, essentially ponto-cerebellar.

6. *Congenital Lesion of the Medulla and Spinal Cord.*—The author presents an extremely interesting case of congenital lesion. It is exceedingly complicated and studied in great detail. It should therefore be considered in the original.

7. *Binocular Vision.*—From the author's studies of many years on experimental lesions of the optic tracts, of the commissures and optic nerves in cats and dogs, etc., he draws the following conclusions: (1) The decussation of the optic nerves at the chiasma is complete in all fishes, amphibia, reptiles and birds, whether possessed of binocular vision or not. (2) Binocular vision is originally associated with carnivorous habits, and is found to a moderate degree among carnivorous fishes, in a few of the sharks and rays, in some amphibia, as the toad, which lives on flies and insects, and in many carnivorous birds, especially the larger gulls, some penguins, hawks, owls and vultures. Among mammals binocular vision is especially developed in the carnivora and in the primates. In the latter group of monkeys and man perfect binocular vision is probably associated with the development of the hand as a prehensile organ. (3) Though many of these animals have a fair binocular vision, yet in all vertebrates below mammals there is total decussation of the optic nerves at the chiasma, and their vision is, therefore, not stereoscopic in the same sense as in the higher mammals, in which visual impressions from the two eyes are received on the same side of the brain, owing to the semi-decussation of the optic nerves at the chiasma; and their macular vision is also less developed than in the higher mammals, as in the felidæ and primates. (4) In animals with laterally placed eyes and periscopic vision the movements of the eyes are independent of each other, as seen typically in the chameleon, while conjugate movements of the eyes for lateral and vertical movements are associated with the acquirement of stereoscopic vision. Convergence of the eyes in the act of feeding is seen in many animals with total decussation of the optic nerves and otherwise independent movements of the eyes, as in the chameleon and the hornbill. (5) The reflex contrac-

tion of the pupil to light is limited to the eye stimulated, and is not consensual, in amphibia and birds, whether possessed of binocular vision or not. In the rabbit, with scarcely any binocular vision, only the one pupil reacts, and the decussation is almost complete. In casts and in the higher mammals with good binocular vision and semi-decussation of the optic nerves at the chiasma there is consensual pupil reaction. Consensual pupil contraction to light is therefore dependent, not on binocular vision alone, but on semi-decussation of the optic nerves at the chiasma. (6) In the rabbit and the cat both the crossed and the uncrossed fibers are spread equally through all parts of the two tracts, and are not connected into any distinct bundle. (7) Experimental division of the optic tract on one side in cats produced distinct homonymous hemianopia, with the hemianopic pupil reaction, and with considerable diminution of the pupil reaction to direct light in the opposite eye, though its consensual reaction was brisk on exposure of the other eye to light. (8) Injuries, either lateral or dorsal, of the anterior part of the anterior corpus quadrigeminum in cats, if deep enough to wound the tegmental roof surrounding the central gray matter, have produced in four cases descending degeneration in Meynert's fountain decussation, most of the fibers decussating at the level of the third nerves to form a longitudinal tract immediately ventral to the opposite posterior longitudinal bundle of the same side. These are the antero-lateral columnar fibers already traced by Boyce into the lower cervical cord. In no case has he produced any degeneration in Forel's lower decussation. (9) In animals with divergent optic axes and periscopic vision and total decussation of the optic nerves there must exist a posterior total decussation of the pupil reflex fibers connecting the anterior quadrigemina with the third nuclei; while in mammals with good binocular vision and semi-decussation of the optic nerves at the chiasma there must be a semi-decussation of the posterior pupil reflex fibers. Consensual reflex contraction of the pupils to light is effected by means of this posterior semi-decussation between the corpora quadrigemina and the third nuclei, and not by means of any commissural fibers between the sphincter pupillæ centers in the third nuclei. (10) The lesion in typical Argyll-Robertson pupil is probably a sclerosis of these fibers, some of the fibers of Meynert's decussation, and simple reflex iridoplegia is not due to a nuclear lesion, though a nuclear lesion may be superadded in the dilated type of Argyll-Robertson pupil, in which the reaction to convergence and accommodation is also lost. **Jelliffe.**

NEUROLOGISCHES CENTRALBLATT

(Vol. 23, 1904, No. 1, February 1.)

1. Two New Skin Reflexes of the Lower Extremity. **Emil Redlich.**
2. Contribution to Myokymia. **J. Meinertz.**
3. Amnesic Aphasia. **S. Popoff.**

1. *New Skin Reflexes.*—Redlich describes two new skin reflexes, the first is obtained by stroking in the mid-line the posterior part of thigh, the leg being held free and in support. There is a contraction of the biceps, semi-tendinosus and semi-membranosus muscles, and sometimes the abductors. The second reflexes obtained by stroking the mid-line of the calf, the toes and foot will be as a consequence in plantar flexion. The reflex is normal, and is diminished in such organic diseases as hemiplegia.

2. *Myokymia.*—The author describes a case of general myokymia in a patient who had other symptoms of a neurasthenic condition. The different theories of the disease are discussed, and the author comes to the conclusion that, as in his case, they are mostly functional in origin.

3. *Amnesic Aphasia.*—Popoff details an interesting history of a man, thirty-one years old, with right hemiplegia and aphasia. He could only say yes and no, had paragraphia, but he had retention of spontaneous

speech. He was not mind-deaf. He could construct sentences, but could not say them unless the words were either spoken first, or else they were written, optic and auditory accessories. Autopsy showed a large lesion, which extended much beyond Broca's area, and involved the second frontal convolution and the whole of the insula and the anterior part of the gyrus temporalis. The author comes to the conclusion: (1) That the motor speech center extends beyond Broca's convolution, and that this additional territory contains a helping center whose function is to help motor speech by optic and acoustic aid, (2) that transcortical aphasia is not dependent on a lesion from B in Lichtheim's zone, but in M (Broca's convolution), and (3) as the patient could write to dictation there is probably a center for agraphia, probably in the second frontal covolution.

(Vol. 23, No. 4, February 15.)

1. Concerning the Localization and the Clinical Meaning of the So-called Bone Sensibility, and the Vibration Feeling. L. Minor.
2. The Relation of the Nervous System to Intelligence. P. Kronthal.
3. Is Unverricht's So-called Familiar Myoclonia a Clinical Entity, which is Established in Neurology? H. Lundborg.
4. Hemiplegia Alternans Superior (Disturbance Relating to the Orientation of the Body). R. Kutner.

1. *Continued Article.*

2. *Nervous System and Psyche.*—In a previous article, which was abstracted in the Journal of Nervous and Mental Disease, the views of the author were stated, regarding the components of the nerve cell. In brief, Kronthal believes that the nerve cells are a mass or collection of leucocytes, which have wandered out of the terminal arterioles of the brain, and have not the functions usually ascribed to them. He then discusses as to what constitutes intelligence. In the protozoa, where there is no nervous system, there is intelligence. The author believes that intelligence is the summation of the different reflexes, and that it does not cause the reflexes. The nerve cells, according to him, therefore would have very little to do with intelligence.

3. *Unverricht's Disease.*—The author discusses the familiar myoclonia first described by Unverricht, and quotes Möbius, who believes that this disease is not a distinct clinical entity, but is simply chronic chorea. Lundborg has had considerable experience in this class of cases, and believes that while there is a resemblance between chorea and Unverricht's disease, the latter is of a distinct type.

4. *Hemiplegia Alternans.*—A patient, fifty-eight years old, suddenly has a slight stroke, which in a few days clears up and leaves the following remaining symptoms: paralysis of the right oculomotor nerve and left facial and hypoglossus. The hemiplegia was left sided. The patient, when he walked, had a distinct cerebellar gait. The patient had different delusions of space. Autopsy showed an area of softening in the right cerebral peduncle near the median line, cutting off some of the fibers of the oculomotor nerve, and also affecting the innermost bundle of the peduncle, thus explaining the cerebellar ataxia.

(Vol. 23, 1904, No. 5, March 1.)

1. A Contribution to the Question of Regeneration of Nerve Fibers in the Areas of Multiple Sclerosis. Bartels.
2. A Reflex of the Dorsum of the Foot. K. Mendel.
3. Concerning the Localization and Clinical Significance of the So-called Bone Sensibility in Vibration Feeling. L. Minor.

1. *Regeneration of Nerve Fibers in Multiple Sclerosis.*—The author discusses the question whether the persisting axis cylinder processes found in multiple sclerosis are persisting or regenerative. Strähuber believes that

they are regenerative. On the other hand, however, Bielschowsky and Hoffmann, by the silver impregnation method, could distinctly trace these axis cylinders, and the author believes, as a result of his work, in the same theories.

2. *A Foot Reflex.*—Striking by means of the percussion hammer the lateral part of the foot in its proximal portion, a dorsal flexion of the second to the fifth toe is seen in normal persons. In organic diseases, where the Babinski reflex is obtained by plantar irritation, dorsal percussion will produce a distinct *plantar* flexion of the other toes. This reflex is therefore of value in differentiating organic from functional nervous disease, for in the later dorsal flexion will always be obtained.

3. *Bone Sensibility.*—Minor discusses the work of Egger and others upon bone sensibility, and comes to the following conclusions: (1) To have a correct comparison of vibration sensation to other forms we should have an uninterrupted vibratory apparatus; (2) the integrity of the bones gives no absolute relation to the condition of the vibratory sensation, for it may not be altered in fractures and dislocations; (3)the vibratory sensation is not altered even in a bone fragment which is proximal to a desired process; (4) Periostitis and caries do not alter this sensation; (5) such alterations as ankyloses and thickenings have no effect upon the bone sensibility; (6) parts of the bony frame, as ribs and vertebræ, which have no sensibility, due to a transverse lesion of the spinal cord, may often respond to bone vibrations; (7) Application of the tuning fork upon the uncovered bone, which is decaying, will give vibration sensation; (8) all these facts lead to the conclusion that the study of the bony sensibility is as yet not definite enough to lead to any conclusions.

(Vol. 23, 1904, No. 6, March 16.)

1. Knowledge of the Mikropsia and the Degenerative Conditions of the Central Nervous System. Dr. Pfister.
2. Concerning a Traumatic Paralysis of the Ramus Volaris Profundus in Ulneris. L. Bregman.
3. Concerning the Muscle Swelling in Myotonia. W. V. Bechterew.
4. Concerning the Therapy of Neurasthenia Sexualis. H. Higier.
5. A Contribution to the Primary Tonic Facial Convulsions, with Muscle Waves. V. Vitek.

1. *Mikropsia.*—The author records the history of an interesting patient who was neither epileptic, hysterical or neurasthenic, but had mikropsia. Such cases are not numerous in the literature. It is thought that the cause is probably toxic, but may run in conjunction with the diseases mentioned. It is directly due to a functional disturbance of the muscular apparatus of the eye. Paresis of accommodation may be present.

2. *Injury of Ramus Volaris Profundus.*—There is no previous record of injury of this particular branch of the ulnar nerve, so the case is worthy of record. The symptoms were as follows: In consequence of the paralysis of the interossii, the first phalanges of the fingers could not be flexed, and the last two could not be extended on account of the contraction of the antagonistic muscles. The adduction and spreading out of the fingers were disturbed. All the distances were more marked in the last fingers. The adductor pollicis and the deep branch of the flexor pollicis nerves were paralyzed. Sensation was not disturbed.

3. *Muscle Swelling in Myotonia.*—Bechterew has observed that by striking the muscles in myotonia the swelling will persist longer than usually, sometimes twelve or fourteen seconds, and that the muscles take from three to five seconds to return to normal form. Deep pressure at times will leave a depression which persists for some seconds. The author considers this observation another symptom of myotonia.

4. *Therapeutics of Neurasthenia Sexualis.*—The author has employed

with success muriate of Heroin in doses of 1 ctg., given nightly in the form of powders, suppositories or pills, in neurasthenia sexual conditions. The dose is decreased gradually, and the intervals are lengthened until patient is cured.

5. *Facial Convulsions.*—Vitex reports a case of muscular contractions of the face with a wave-like motion of the muscular fibers. Bernhardt reported the first case, and Unverricht the second. The author assumes that the cause is central (Spiller reported a similar instance before the Philadelphia Neurological Society, so this new case really makes the fourth instead of the third.—*Abst.*). WEISENBURG.

REVUE DE PSYCHIATRIE ET DE PSYCHOLOGIE EXPERIMENTALE

(May, 1904.)

1. The Hypochondriacal Idea. MARCHAUD.
2. The Disorders of the Ocular Reflexes Studied in the Same Patients at the Three Periods of General Paralysis. MARANDON DE MONTYEL.

1. *The Hypochondriac Idea.*—Normally man is not conscious of his several organs. The sensations from them do not cross the threshold of his consciousness. Consciousness of organic sensations results either from an increase in the intensity of the sensations originating from the internal organs, or as a result of directing the mind especially on them. Both states are abnormal, the former giving rise to symptomatic hypochondria, the latter to essential hypochondria. At the base of hypochondriacal ideas there is an emotional state of depression. The hypochondriac is in a continual state of depression. Their sad ideas relate to their personality, to imaginary troubles in the functioning of their organs, are auto-accusatory. Closely allied to the hypochondriacal ideas are the ideas of negation. In the former the patient suffers from imaginary troubles with his organs, in the latter he believes he has no brains, stomach, etc., as the case may be. The two sorts of ideas are often found in the same patient, and the hypochondriacal ideas may give place to the ideas of negation. With reference to their differentiation from ideas of persecution it may be said that the hypochondriacal ideas are auto-accusatory, while the persecutory ideas are allo-accusatory. The persecuted does not accuse his organs of making him suffer; the cause of his suffering is external to his body. The hypochondriacal idea could then be defined as an idea with auto-accusatory tendencies provoked by a sad emotional state, without characters of negation, and relating uniquely to the personality of the patient. The hypochondiacal idea is important for diagnostic purposes. Taken alone it is not sufficient, but it is always accompanied by reasoning, more or less logical, which permits of an appreciation of the intelligence of the patient. As a rule hypochondria develops only in brains that have been poorly developed, and is only rarely due to a peripheral trouble in some organ. There are three classes of hypochondriacs. Those suffering from hypochondriacal preoccupation, who complain in a more or less reasonable way, and for the most part are never confined; those with hypochondriacal delirium, whose ideas are most absurd, such as a belief that the brains are decomposed, etc.; and those with fixed hypochondriacal ideas. These stand in distinction from the first two classes in which the ideas change from day to day.

Here follows a tabulated analysis of 695 cases—360 men and 335 women. Of this number of patients examined, fifty-two preesnted hypochondriacal ideas. Hypochondriacal ideas are shown to be most frequent in conditions of dementia, particularly paralytic dementia, but, on the contrary, are rare in idiocy and imbecility. They are associated very frequently with melancholy ideas, but rarely, as would be expected from the definition, with ideas of grandeur and states of exaltation, only one such case appearing in the tables. Of forty-five patients who had attempted suicide eleven had hypochondriacal ideas. Among hysterics they are very rare, no case appearing in the author's studies.

2. *Ocular Reflexes in Paresis.*—This is a synthesis of three preceeding studies by the author on the conjunctival, accommodation and light reflexes not in accordance with the usual method of studying the conditions in different patients in the different stages of the disease, but by continuous observations on the same patients throughout the course of their malady. The article is already so condensed as not to bear abstraction.

(June, 1904.)

1. The Delirium of Interpretation. Sériex and Capgras.

1. *Delirium of Interpretation.*—"The delirium of interpretation is a perfectly defined clinical species characterized by the following signs: Development of systematized delirium of diverse formulas; frequent absence (or rôle always secondary) of hallucinations; extreme richness of the interpretations, substratum of delirium; very slow, progressive march, without systematic evolution and without terminal dementia. The delirium of interpretation should be distinguished on the one hand from the classical delirium of persecution with preponderance of sensory disturbances, and on the other from the insanity of the persecuted—persecutors."

It is proposed to study successively: 1. The content of the delirium. 2. The delirious interpretations. 3. The episodic hallucinations. 4. The mental state. 5. The varieties. 6. The evolution. 1. *Delirium*—The particular form of the delirium is of no importance. It depends on the intellectual level of the patient, his education, whether he is naturally persecuted, mystical, etc. Ideas of persecution are often found, simple or combined with ideas of grandeur, much as in the systematized hallucinatory psychons; but the intellectual integrity does not permit them to so pass the bounds of the reasonable. Ideas of jealousy and certain forms of hypochondria are found. Ideas of grandeur are frequent, and finally mystical and erotic ideas conclude the list of those ideas that have anything characteristic about them. 2. *Delirious Interpretations*—Whatever there is of color and richness to the delirium is furnished by the interpretations. It is interesting to know the point of departure, the provocative agent of these interpretations. At first the patient is ingenious. Often a word is thought to have some mysterious significance each time it is pronounced in conversation. A gesture or some circumstance is falsely interpreted. Then the patients pass in review their life history, and receive confirmation for their ideas from many things remembered. Organic sensations, often perfectly normal ones, add ideas, and finally a complete change of personality of the patient occurs, and often of all those about him. He spells his name differently, adopts an illustrious name, does not recognize relatives, etc. 3. *Hallucinations*—Hallucinations are rare, episodic, transitory, without progressive evolution, and have only a secondary influence in the course of the psychons. This is in marked contrast to the part they play in chronic delirium. 4. *Intellectual State*—Perfect lucidity with a remarkable power of reasoning is retained throughout. There is present hypermnesia for details, and proof upon proof is accumulated. These patients are often graphomaniacs, and with multitudes of letters, books, memoirs, etc., the diction of which is perfect and free from cabalistic signs and exaggerations. Because of their lucidity and eloquent persuasiveness it not infrequently happens that they communicate their convictions to those about them. This often happens to near relatives, as mother and daughter, brother and sister. 5. *Varieties*—The cases may be classed as political persecution, hypochondriacs, erotic, etc., or better simply divide them into active and passive. The former become persecutors, the latter are mostly persecuted megalomaniacs, inoffensive, deteriorated and good natured. 6. *Evolution*—The evolution of the delirium of interpretation presents three essential characteristics: (1) Chronicity; (2) progressive but not systematical evolution; (3) absence of terminal dementia. When these cases are admitted to the hospital their delirium is usually in full bloom, but they are reticent, and often present a retrospective delirium, which often leads to the false diagnosis of organic paranoia. The absence of terminal dementia is an important characteristic. The delusional system retains its integrity to the end unless it may suffer some as a result of senility.

As to etiology, about all that can be said is that the disease is built upon a congenitally defective brain. The principal conditions which may be confounded with the delirium of interpretation are: the insanity of the persecuted persecutors, the delirium of persecution with hallucinatory foundation, and certain psychons, with a preponderance of delirious interpretations, particularly toxic conditions. The prognosis is absolutely unfavorable. Only senile involution and not the evolution of the psychons will, however, lead to enfeeblement of the faculties.

W. A. White (Washington.)

CENTRALBLATT FUER NERVENHEILKUNDE UND PSYCHIATRIE

(Vol. 29, 1904, No. 8, August.)

1. Notes on the Anatomic Bases of Idiocy. Dr. Alzheimer (Munich).
2. L. Lowenfeld's Book on the Psychic Impulsive Manifestations. M. Friedmann.
3. Contribution to the Knowledge of the Flight of Ideas in Mania. A. Schott.

1. *Pathology of Idiocy.*—",Secondary dementia" has played a great rôle in psychiatry; its conception is based not on the cause, the mode of development, or the character of the insanity, and thus it may be compared with the conception of idiocy, which is simply a defective mental state existing at birth or acquired in early years. Ballet thinks idiocy is an old, bad word; it comprises a number of pathological entities. Besides Cretinism, which is placed by Kraepelin among the diseases of metabolism, there are Microcephaly and Macrocephaly, Micro- and Macrogyria, Porencephaly and Hyrocephaly. But the causes of these conditions are diverse disease-processes affecting the developing brain. The changes of paresis in the adult brain are hard to distinguish from the changes in the brains of some idiots; is there possibly a congenital paresis? Probably not; but Alzheimer is convinced that some juvenile paretics are to be found in every large idiot asylum, and he thinks many of Bourneville's "meningitic idiots" are juvenile paretics. After giving an acount of amaurotic family idiocy, Alzheimer declares that here is a disease to which none in the adult can be compared. Hypertrophic tuberous sclerosis (Bourneville) does, judging from Furstner's cases, occur later than childhood. Often in these cases focal hyperplasia of neuroglia is found. Encephalitis is one of the most common causes of idiocy; it occurs in patches whose favorite seat is at the junction of gray and white matter. These leave areas of atrophy, amounting sometimes to porencephaly, and causing symptoms according to their seat. In addition to syphilitic endarteritis, emboli, traumatic hemorrhages and injuries of the brain, especially at birth, tumors may induce idiotic states. The old idea was that idiot brains were simply brains checked in their development, while idiots belonged to different grades according to the stage at which this checking took place. Alzheimer combats this on the ground of the various pathological conditions so generally met with. And, moreover, like Kraepelin, he believes that certain idiotic states are really dementias, early instances of dementia præcox, with marked katatonic traits. Alzheimer laments the fact that in Germany the idiot asylums are still under non-medical care; this keeps back the study of this class of

mental affections. It is to be remembered that idiocy is used above in the generic sense to include imbecility.)

2. *Löwenfeld's Book on the Impulsive States.*—Friedmann (Mannheim) says that each author classes the impulsive insanities in his own way, and as Mendel puts it, can, with the help of a Greek lexicon, continually foist new forms upon us. Krafft-Ebing and Westphal were the first to describe the "Imperative-conception-disease" (Zwangsvorstellungskrankheit); then its relation to neurasthenia was pointed out; next Magnan arged the influence of hereditary degeneration; Freud conceived an origin trom sexual perversions or errors; others have emphasized the disorder of will in these cases. Friedmann has described impulsive states at puberty and at the climacteric, also a "neurasthenic melancholia"; Löwenfeld has adopted the division of impulsions into intellectual, emotional and motor, and he makes minute sub-divisions to embrace all cases. Friedmann considers this classification, compares it with others, and studies the subject psychologically.

3. *Flight of Ideas in Mania.*—Schott, of Weinsberg, contests the idea that in mania there is mere motor excitation with preserved power of attention. The latter, he says, is disturbed, as shown by the patient's diminished power of concentration; and side by side with the psychomotor exaltation there is a psychosensory exaltation which may at times be more prominent than the motor.

(Vol. 29, 1904, No. 9, September.)

1. On the Question of Stationary Paralysis. C. WICKEL.

1. *Stationary Paralysis.*—We may speak of "stationary paresis" in a sense when remission or intermission occurs in the course of this disease; but in true stationary paresis there is no abatement or disappearance of symptoms, only a standstill at some stage for years. These several phenomena account for the slow course of some cases of paresis. Binswanger reported cases lasting twenty years. Tabes-paralysis and paresis combined with disseminated sclerosis tend to this long course. Gaupp and Alzheimer have doubted the genuiness of such cases, the former calling them "paresis-like dementia." Wickel reports three cases which were stationary for eight and one-half, seven and one-half and five and one-half years respectively. He considers the diagnoses of brain-syphilis, alcoholic and traumatic dementia, and excludes them.

(Vol. 29, 1904, No. 10, October.)

1. Old Dementia Præcox. W. WEYGANDT.

Dementia Præcox.—In the Julius Hospital, says Weygandt, insane cases have been kept from its foundation in 1576, making it the oldest psychiatric establishment in Germany. This asylum contains patients who were admitted as far back as 1850, and though they have merely vegetated there, and were for a long time designated "idiots," yet a comparison of their early with their present state is interesting. The insanity in these old cases began in youth with hallucinations, absurd delusions, strange actions, mannerisms, neologisms, etc., leading to a peculiar dementia. At present in all these cases there is a simple want of spontaneity; the memory is intact. One of four patients shows confusion of speech; another has been somewhat cataleptic during fifty years. A striking feature is the absence of senile dementia in these cases. The defect in these dements is in the realm of apperception and of will (in the sense of Wundt), as a result of which also feeling is reduced. Weygandt considers farther the psychology of these states.

PICKETT (Philadelphia.)

AMERICAN JOURNAL OF INSANITY

(Vol. 61, 1904, No. 1.)

1. Suggestions and Plans for Psychopathic Wards, etc. L. P. Clark and H. P. Montgomery.
2. Syphilis of Nervous System. E. W. Mooers.
3. Medico-legal Case of Well Poisoning. H. R. Stedman.
4. Blood Pressure in the Insane. W. P. Dunton, Jr.
5. Characteristics of the Scotch Lunacy System. Owen Copp.
6. Astasia Abasia Associated with Epilepsy. W. H. Buhlig.
7. Colony Building for the Defective Classes. W. P. Spratling.
8. Status Epilepticus. L. P. Clark and T. P. Prout.
9. Influence of Mode of Life on the Blood of the Inmates of a Hospital for the Insane. W. G. Melvin.

1. *Suggestions and Plans for Psychopatic Wards, etc.*—Some suggestions, based mainly upon a visit to some of the more recently constructed German psychopathic wards and hospitals, notably the Psychiatric Clinic at Kiel, with a number of very instructive plans, by Mr. Montgomery, an architect.

2. *Syphilis of the Nervous System.*—A case, from the Maclean Hospital, of a man of forty years old, who showed an amnesic symptom-complex, with gradual dementia and certain focal manifestations, the latter being fairly well accounted for by the autopsy findings. The pathological changes consisted in meningeal thickening, arterial disease, neuroglia proliferation, cell degeneration and local gummatous deposits. The details of cell cannot be entered into here.

3. *A Medico-legal Case of Well Poisoning with a Plea for a Hospital Observation Law.*—An account of the case of a man accused of the above crime, who, only after repeated examination, was decided to be a defective and mentally irresponsible.

The difficulty of decision in such cases has led the author to make a plea for a law authorizing the commitment to a hospital for the insane of persons accused of crime, about whose mental condition there is a doubt, in case the medical examiners decide that a prolonged observation is necessary to resolve such doubt. Upon his initiative the Boston Society of Psychiatry and Neurology appointed a committee, who succeeded in securing in Massachusetts legislation authorizing the court when necessary to order such detention. A similar statute exists in Maine, Vermont and New Hampshire, and is found to work well.

4. *Blood Pressure in the Insane.*—The author has studied the blood pressure by means of a modified Riva-Rocci syhygmomanometer, in twenty-five insane women of various ages, and presenting various mental conditions, numerous observations being made. extending over periods of several months. He has not so far found this determination of much practical assistance from a diagnostic point of view, but thinks the subject worth more extended investigation. Upon his studies he bases the following conclusions: 1. The findings of other writers that blood pressure is increased in depressive states, and decreased in excited states, and that the motor condition has a greater influence on the blood pressure than does the mental condition have been confirmed. 2. "A moist skin has no special influence upon the blood pressure though active perspiration may." 3. There is no constant variation, as has been shown by Schaeffer and others.

5. *Scotch Lunacy System.*—Owen Copp outlines the general method and tendencies in the care of the insane, observed by the author during a recent visit to Scotland. The salient features of the Scotch system the author epitomizes as follows: 1. "The hospital idea embodied at present in the reception hospital, which, in urban localities, seems likely to expand

into the psychiatric clinic." 2. "The colony idea expressed at existing asylums in farmsteadings, industrial groups, home villas and outlying farms, and in the new colonies projected after the Alt-Scherbitz plan." 3. "Community care now facilitated by boarding in families and temporary treatment in unlicensed houses by a general practitioner."

6. *Astasia-Abasia Associated with Epilepsy.*—Description of a case showing the above symptom, together with certain other hysterical manifestations, and having besides from time to time undoubted attacks of grand mal.

7 *Colony Buildings for the Defective Classes.*—The colony plan may serve equally well for epileptics, the insane, the feeble minded, and for a proportion of the reformatory classes. Basing his opinion upon his experience at the Craig Colony for Epileptics the author makes the following suggestions: A large tract of land should be secured, an acre per patient is not too much. The buildings should be arranged so that starting out from the administration building, hospital, etc., the residences for the best patients come next, the intermediate class are placed further off, while farthest away are located the irresponsible, infirm and generally objectionable. He illustrates his meaning by a diagram of concentric circles—though he expressly states that the buildings are not of necessity to be built in concentric rings. The inner and smallest circle is to contain the office building, hospital, laboratory, chapel, laundry, schools, storehouse, industries, library and employment homes. In the next circle come the houses for the best class of patients (at Craig twenty per cent. of the total), each for from sixteen to eighteen patients, half of whom have single rooms. In the third circle are houses for twenty-eight to thirty persons, two to five in a room, the intermediate class (at Craig sixty per cent.), while the outer circle is for the bedridden, infirm and chronic insane, 125 to 150 to a house (at Craig twenty per cent.).

8. *Status Epilepticus*—(Concluded from Vol. 60, Nos. 2 and 4).—L. Pierce Clark and Thomas P. Prout base this clinico-pathological study on thirty eight patients with status, five cases of serial attacks ("état mal de passage"), and four cases of status equivalent. The authors at the start give the following definition: "Status epilepticus is the maximum development of epilepsy, in which one paroxysm follows another so closely that the coma and exhaustion are continuous between the seizures." Status is sooner or later accompanied by rise of temperature, and rate of pulse and respiration; in the great majority of cases the extent and character of this rise giving important prognostic indications. Status occurs mainly in connection with grand mal epilepsy, hence it is chiefly characterized by convulsions, either general or local, followed by exhaustion and coma, but the various epileptic equivalents occur. The spasm may be tonic or clonic, or of one form only. Status occurs mainly in "idiopathic" epilepsy, but is a notable ending in Jacksonian and symtomatic forms. The authors consider it the natural climax of all cases of epilepsy not carried off by some intercurrent disease. They cannot find that age, sex or menstruation exercise any special influence in its determination. As to time elapsing between the establishment of the fits and the first attack of status, an average in twenty-three cases was found to be twelve years. The epilepsy must become well established before status supervenes. The intervals between the attacks of status are very irregular. As a rule, the second or third period will prove .fatal in "idiopathic" epilepsy, though the authors have seen cases surviving four, five and six attacks. The further short of typical grand mal attacks the individual seizures fall in severity, the less the gravity of the status. As a rule status is heralded by increased frequency of paroxysms, a history of serial attacks rising from five to fifteen, or twenty in twenty-four hours has been the rule. Serial periods of ten to twenty seizures, attended by slight rise of temperature with but little

prostration, have been called by Bourneville "état de mal passage." At the onset the grand mal attacks usually occur at intervals of a half hour to an hour. In partial epilepsy "the single seizure of the status holds to a distinct order of invasion as long as exhaustion is not extreme, and the definite order of muscular involvement is continued throughout the status." At first consciousness is regained between the seizures, but as they grow more frequent the patient becomes stuporous, then profoundly comatose. The temperature rise reaches in some cases 107° or 108° F., the pulse rate mounting to 160 to 200 per minute. At last the convulsions lessen in frequency, and the patient lies in the coma of collapse. He may die in the convulsion, but as a rule he passes a few hours in coma, which is either followed by death or gradually passes into stupor, then into mild delirium, which slowly proceeds to recovery, though if this is long delayed extensive sloughing about the nates takes place, and the patient dies. There may be from fifty to two hundred seizures a day, six of the authors' cases having been between 100 and 200. Partial attacks are much less serious, one of the cases having seventy-five in a day without much prostration, while another had 755 absences in twelve hours. As a general rule the convulsive stage lasts from eight or ten hours to three or four days. The stuporous and comatose stage is largely the product of the convulsive stage. It lasts for varying periods, in the majority of instances from two to nine days. The authors devote some space to the consideration of transitory hemiplegias and other paralyses following repeated seizures and status, which they regard as being due to exhaustion of the cerebral centers. The reflexes are lost in the comatose stage, and exaggerated after it, remaining so for weeks or months. Salivary secretion is arrested in the course of status, and the tongue, lips and cheeks become dry and fissured. There is gradually paralysis of the pharyngeal muscles, the throat becomes filled with mucus, and swallowing is impossible. Epistaxis and hemoptysis have been noted. The pupils are usually dilated, but may show various irregularities. Subconjunctival hemorrhages are frequent. The skin in the later stages is cold and clammy. It sometimes shows erythematous, urticarial or herpetic eruptions. Subcutaneous hemorrhages are frequent. Among trophic disorders the most remarkable are bed sores and loss of weight. The urine—which together with the feces is often voided involuntarily—may contain albumin, blood and casts. The sweat is very abundant, but detailed studies upon it and upon the blood seem to be wanting. The study of the fever, pulse and respiration curves gives important prognostic indications, their continued rise being a signum mali ominis. The authors enter into a discussion of the cause of the fever, which cannot be detailed here. The diagnosis of status depends chiefly upon the history of previous epilepsy, and in the absence of this may present some difficulty, though close attention to symptoms will, as a rule, point out the distinction between it and apoplectic and uremic states, etc. The prognosis of status, while very grave, is not necessarily hopeless, the mortality running from thirty-three to fifty per cent. Calculating this, however, upon the whole number of status periods, instead of upon the number of patients (which the authors think would be more rational), they find it fourteen per cent. only. The advance of the symptoms unmodified by treatment points to a fatal issue. Into the authors' discussion of the pathology of epilepsy in general it is impossible to follow here. The pathological findings, both gross and microscopical, are next considered, many of those reported being put aside as irrelevant. It is well known that in epileptics many abnormalities in skull, brain and membranes are found. These the authors are inclined to look upon as developmental defects, in which category they would also place the thymic hyperplasia, and evidences of lymphatic diathesis described by Ohlmacher. In their microscopical investigations they have proceeded upon what they consider an established fact, that epilepsy is a disease of the cortex, and have studied especially the cortical cells and the neuroglia. Seven cases of status were examined, and the findings compared with those in twelve other epileptics. The changes found affect chiefly the cells of the second, and to a less extent, those of the third layer of the cortex. They seem to begin in the nucleus and lead to disappearance of the nuclear membrane and the karyoplastic reticulum, favoring displacement of the nucleolus. The chromatic substance of the cell also disappears, and at length only a dimly outlined cell body remains, many neurones seeming to disappear. The cortex is invaded by leucocytes, and there is neuroglia proliferation. Considering the meaning of these changes the authors regard as characteristic the pre-eminent involvement of the nucleus, in which they think the formative power of the cell centers, and which is the essential element in chemical synthesis. They draw the following conclusions: 1. "If the especial involvement of any particular type of cell is indicative of the essentially sensory or motor character of epilepsy, it would seem that Prus was correct in concluding that epilepsy is essentially a sensory phenomenon, as the cells of the second layer and the third layer, in lesser degree, are especially involved." 2. "The essential lesion of epilepsy pertains to the nucleus of certain of the cortical cells, and is of such nature as to seriously jeopardize the cell for considerable periods and ultimately cause its destruction." 3. "The chromatolysis in epilepsy is a nutritional change, brought about by the nuclear toxemia, since the nucleus presides over the process of elimination, absorption and digestion in the cell unit." 4. "The rôle of the leucocyte in the cortex, after severe epileptic explosions, is most probably that of a phagocyte." 5. "The neuroglia overgrowth in epilepsy is one of its more remote sequences, and probably occurs in response to toxic irritation. The treatment of status must be largely prophylactic, though it cannot always be prevented. The tendency to serial attacks should be reduced by bromides. For incipent status the following is recommended:—

℞—Tinct opii deodorat	m V
Potass brimid	gr. XXV
Chloral hydrat	gr. XX
Liq morphin sulph (U. S.)	dr. I

Until this can take effect the convulsions should be controlled by chloroform. Venesection may have a place in the few plethoric cases, but as a rule it is best followed by saline injections. After status is well established chloral and bromide, by rectum, and morphia, hypodermically, may be called for. A number of other drugs are discussed, but the authors consider them of doubtful utility. Bromide of sodium in ten per cent. solution may be given hypodermically, but generally leads to abscess. The stuporous stage requires supportive treatment with special attention to nutrition and stimulation. Peptonized milk, with eggs and beef extract, should be given, and tube feeding may be required. That the patient needs plenty of water should not be forgotten, and enteroclysis, or hypodemoclysis, may be indicated. For the restlessness and sleeplessnes of the stage of delirium, the milder hypnotics will be, as a rule, found sufficient.

9. *Influence of Mode of Life Upon the Blood of the Inmates of a Hospital for the Insane.*—The author made blood counts and hemoglobin determinations in fifteen patients of each of the following classes: 1. The newly admitted. 2. Chronic patients remaining mostly indoors. 3. Chronic patients working out of doors. 4. Patients living in tents on special diet without medicinal treatment. In Class 1 the Hb. averaged 60-70 per cent., in Class 2, 50-70 per cent., in Class 3, 70-90 per cent., and in Class 4, 80-90 per cent. The number of red cells closely paralleled the Hb. His results with diagnoses of mental diseases and remarks are exhibited in tabular form.

Allen (Trenton.)

JOURNAL DE NEUOLOGIE

(Vol. 9, 1904, Nos. 15 and 16.)

Devoted exclusively to a report of the proceedings of the Fourteenth Congress of Alienists and Neurologists of France.

(Vol. 9, 1904, No. 17.)

1. A Case of Chronic Progressive Chorea with Autopsy. D. De Buck.

1. *Chorea.*—Report of a case of chorea with dementia in a man of sixty-five years old, followed by autopsy. Of the changes found in the cortex of the brain, neuroglia proliferation and cell degeneration, with diminution of the tangential and supradiar fibers was most marked. The author ranges himself upon the side of those who consider the pathological process in chronic chorea, a sclerosis and not an inflammation. This sclerosis he thinks secondary to the degenerative changes in the nerve cells.

Allen (Trenton.)

ARCHIVES DE NEUROLOGIE

(Vol. XVII., 1904, No. 102, June.)

1. A Case of Dementia Præcox of Irregular Type. R. Masselon.
2. Delirium of Persecution in Progressive Paralysis. Pasturel.

1. *A Case of Dementia Præcox of Irregular Type.*—The author gives a detailed history of a most excellently observed psychosis. The patient was a man of brilliant intelligence up to the age of twenty-four, when he developed an accession of delirious type, which was followed by a condition of intellectual enfeeblement, which was by no means an extreme dementia. The principal features of interest were the dominant state of aboulia and general indifference. An individual, formerly very capable and gifted, sinks to the level where he prefers asylum life and shuns with dread every intellectual effort. There was no emotional suffering, no depressive state that was painfully appreciated as in melancholia, nor was there any element of neurasthenic or a hypochondriacal nature. The subject simply presented an aboulia, an emotional indifference, a diminution in the vividness of his mental concepts. There were lacking the negativism, stereotypy, or complete mental reduction, but Masselon regards these as but manifestations of an early weakening of the intellect. Negativism, stereotypic or catatonic attitudes represent extreme forms of precocious dementia. In his case the patient never *spontaneously* thought of anything, or nearly so, but one could make him use his mental powers momentarily by directing his thoughts in a certain direction, or by artificially sustaining his attention. Masselon follows Aschaffenburg in creating a class of cases to be grouped under the heading of "formes frustes" of dementia præcox. Aboulia and mental apathy with lack of spontaneity and initiative are the special features. The clinical description laid down by Kraeplin is thus modified to include cases of milder type, which were formerly otherwise classified.

2. *Delirium of Persecution in Progressive Paralysis.*—After commenting on the rarity of systematized ideas of persecution in general paralysis, the author states that he had opportunity to study three cases which showed persecutory delirium of great tenacity and extraordinary intensity. These ideas are usually accompanied by visual and auditory hallucinations (Ballet). Pasturel's cases were dangerous to the personnel of the institution. Ideas of persecution when associated with paresis are considered by Baillarger as a new malady superimposed. Ball views them as a complication. Magnan and his school considered that their presence in paresis implied a vesanic or neuropathic heredity, or alcoholism. Troget laid great stress upon the latter factor. Others attributed these delusions of persecution to a localized meningo-encephalitis. Sérieux reported an interesting autopsy with meningitis at the level of the supra marginal and posterior third of

the first temporal convolution, where there were auditory hallucinations with ideas of persecution and jealousy. Pasturel records his observations carefully and is inclined to agree with Sérieux that general paralysis engenders by its irritative lesions, along with its multiple hallucinations, persecutory ideas and persistent violent reactionary states. Heredity and alcohol are often assisting factors. D. I. Wolfstein (Cincinnati.)

MISCELLANY

Hypnotic Remedies in Mental Disease. A. R. Diefendorf (Journal of Amer. Med. Asso., November 19, 1904).

The paramount importance of insomnia as a symptom of mental disease makes the indications for hypnotic remedies foremost in the therapeutics of psychiatry. Of the two types of insomnia, (1) insomnia of the early stages of mental disturbance; (2) the insomnia encountered after the psychosis has become well established, the first is very frequently intense, and preceding the other more active symptoms sometimes many months. It may be associated with peculiar sensations, such as a feeling of weight and construction, or with a certain reserve and solitariness or an irritable mood. It may be associated with distressing dreams. There is no specific remedy, but exciting causes must be removed, and gastric, cardiac, naso-pharyngeal irritation be treated, or altered constitutional states, such as lethema, gout or anemia improved. Hypnotic drugs should be the last resort; warm bottles or other hydrotherapeutic measures should be used. The writer advocates bromides, especially bromopin, or trional or veronal. The insomnia of well-established mental disease is usually less urgent, and is apt to be associated with hallucinations or fears. Delirium tremens calls for hot packs and choral, or, perhaps paraldehyde or chloralamid. If there is difficulty in getting the patient ready for his first hot bath, give hyosin hydrobromate, or scapolamin. Trional or veronal are less reliable. The insomnia of the depressive phases of dementia præcox and mania depressive insanity are best ameliorated by cold or warm pack, choral and bromides in combination. For the extreme insomnia of exhaustion and infecion phychoses, alcohol is most valuable. One of the most obstinate forms of insomnia is in senile dementia.

Noyes (New York.)

Collargolum in Nervous Affections.—At the meeting of the Philadelphia Pediatric Society, Dec. 8, 1903, Dr. D. L. Edsall reported a case of chorea of the septic type treated with intravenous injection of collargolum, which apparently had a markedly favorable effect upon the temperature and a striking effect upon the child's general condition. The general septic appearance of the patient, together with the prostration, rapidly disappeared. The choreiform movements, also, soon improved greatly, although for a week previously the child had been ineffectively treated with sedatives, salicylates, etc. She ultimately recovered entirely, except for a gravely damaged heart. The speaker also referred to other cases, partly in children, partly in adults, in which he had treated rheumatism, septis, typhoid fever and other conditions with intravenous injections of collargolum. He had seen no bad effects, except in one or two instances in which sore arms had been produced by missing the vein and injecting collargolum into the subcutaneous tissues. As to the influence of collargolum upon the infections in the various cases, he was disposed to be guarded in his statements; but he felt that there had been a favorable influence exerted in a number of cases of septicemia, and that the effects had been sufficiently marked to encourage him in using the preparation further.

To six epileptics Netter (*Bull. des Hop.* April 28, 1904) administered collargolum in liquid and pill form in conjunction with the usual treatment. and found it very advantageous. The specific action of the latter drug was

gotten from much smaller than the usual doses; and the distressing symptoms of bromism, the eruptions, bad breath and mental depression were avoided. He has even used collargolum alone in several cases and in a number of instances has had patients pass five or six months without an attack.
G. GOODALE (New York.)

INDIRECT ATROPHY OF CEREBELLUM Dr. Reitsema (Psychiatrische en Neuroligische Bladen, Vol. 9, 1904, No. 3, May, June).

About the influence of a cerebral defect on the growth of the contralateral cerebellum, there exists a great deal of controversy. In normal individuals there is no constant relation between the weight of the cerebrum and cerebellum, and cases are not rare of large cerebral defects without perceptible changes in the cerebellum. The author relates a case of nearly absolute absence of the left hemisphere and a very pronounced diminution of the contra-lateral cerebellum. In this case the red nucleus was strikingly intact, whereas the superior cerebellar peduncle was very reduced. Contrary to the opinion of the authors, who deny that the cerebellum atrophies after cerebral defects, or regard this atrophy as a second degree atrophy, or will explain the cerebellar atrophy, purely mechanically. The author does not believe in the possibility to show that the intensity of the cerebellar shrinking be parallel to the size of the cerebral defect. Nor does he think that the cerebellar atrophy is dependent on destruction of special parts of the cerebrum. Further, he regards the cases of homolateral atrophy of cerebellum and cerebrum (Virchow, Kundrat, Kahlden) a great difficulty. He expects something from experimental work.
MUSKENS (Hague.)

THE BRIDGING OF NERVE DEFECTS. Charles A. Powers (Annals of Surgery, November, 1904).

Although correction of the evils resulting from a gap in the continuity of a nerve is a matter of great importance in a given case; it hardly seems possible at this time to say definitely what form of bridging should be employed. More cases, and especially cases reported later and better, are needed. Neuroplasty and implantation (anastomosies) are always available resources, and for the present it would seem that they should be preferred. Resection of bone may be advisable in selected cases. Transplantation of foreign grafts should be abandoned. It is hardly necessary to say that prognosis in an individual case should always be guarded, and that repeated operations may be necessary.
(NOYES.)

PSYCHOSES AND BRAIN TUMORS. J. van der Kolk (Psychiatrische en Neurologische Bladen, Vol. 9, 1904, No. 4, July, August).

The author relates a case of a man, forty-three years old, married, and father of six healthy children, gardner; treated in the asylum for four years, giving no appreciable symptom of cerebral tumor. As an inert mass he was laid up, was incontinent, showed continually a low degree of negativismus; no hallucinations. The pupils were unequal, reacted well; there was a slight facial paresis on the right side, the knee-jerks were exaggerated. There was no progressive dementia. The autopsy showed a great number of tumors in the medulla oblongata, one in the former part of the left temporal convolution, one in the left gyrus lingualis, and one in the right gyrus parietalis. After microscopical examination they appeared to be cavernous angiomata. In the literature no cavernous angiomata are related as a cause of psychosis. The only cases the author found are one of Shover, which gave rise to epilepsy, and one of Ohlmacher. In the last case as well as his own the author does not think that the psychical symptoms could be caused by direct mechanical lesion of cerebral regions involved in the psychical life. Moreover, cavernous angiomata do not cause compression, nor do they infiltrate the tissue. He thinks that after Pierre

Marie cerebral edema caused the symptoms. Equally he regards the psychical symptoms caused by toxic polyneuritis, caused by edema gravidity psychosis; abortus provocatus; recovery. H. Treub relates the case of a young woman, thirty-one years old, who had from the beginning of the gravidity hypochondriacal ideas. She was convinced that she would give birth to a monster. She made several attempts at suicide. She would not take any food, in order to prevent the monster from growing. Although generally psychiatrists do not expect any effect from abortus, in this case Winkler and Treub decided to do abortus provocatus with laminaria. Within two weeks she completely recovered.

MUSKENS (Hague.)

TRAUMATIC ABSCESS OF CEREBRUM. Ernest F. Robinson Annals of Surgery, November, 1904).

A case of attempted suicide produced the following results: A wound from a 32-caliber revolver behind the right ear, shattered the petrous portion of the temporal bone. The patient survived, and paralysis of the right side of the face, and inability to close right eyelids, due to injury of the seventh nerve, persisted. Six weeks after the injury she was found to be suffering from frightful headache over the right side of head, forehead and eye. Her mental faculties were decidedly dull or delayed. She was mildly delirious at times. Her temperature was subnormal, and her pulse was slow but weak, at 52 per minute. The pupils were markedly unequal, the right dilated, but reacted slowly to light. Nystagmus was not present. No changes in the fundus could be detected. The patient had vomited. The tongue deviated markedly to the left side. An abscess of the temporosphenoidal lobe had developed, either from an infection or from devitalizing an area of brain substance. Operation and aspiration discovered pus, which was drained, and a tube left in for a short time. Recovery was uneventful and rapid.

NOYES (New York.)

FRACTURE OF THE BASE OF THE SKULL. George L. Walton (Annals of Surgery, November, 1904).

1. In the majority of the cases the basal fracture resulted from impact received in the horizontal plane of the skull, whether upon the frontal or the occipital region or upon the side of the head. 2. While certain of the basal fractures extended from the vertex, there was no suggestion of the *contre-coup* of earlier writers. 3. The line of fracture tended to enter the fossa nearest the point of impact, and to extend in the general direction in which force was applied. 4. The lines of fracture in traversing the base tended to follow lines of least resistance, and in twenty-two of the fifty cases these lines corresponded more or less accurately to those indicated by Rawling, but the exceptions were too marked and too constant to allow the establishment of fixed rules. 5. The sella turcica was implicated in thirty-six per cent. of the fractures. The petrooccipital and masto-occipital sutures furnished lines of least resistance. Fractures extending across the base tended to run parallel to the petrous portion of the temporal bone and through the sella turcica. Certain blows on the occiput tended to cause a line of fracture extending to the jugular foramen or across the petrous bone. The portion of the petrous bone containing the auditory apparatus showed itself peculiarly liable to fracture, more often transversely than longitudinally. 6. In seven cases (fourteen per cent.) the fracture was limited to the base after vault impact in the horizontal plane. Neither Rawling's theory of transmitted force nor the theory of bursting fracture of von Wahl and others suffices alone to explain these cases. The results of experimenting with bodies of simpler structure would suggest that the bursting principle predominates in pure compression of the skull, and the principle of transmitted force in case of blows, while both play important

parts in case of falls. 7. The orbital foramen was implicated in 21.4 per cent. of the cases of orbital fossa fracture. 8. Inequality and immobility of pupils, or both, furnish the most frequent and unfavorable sign of fracture of the base. In the forty-four cases in which the pupils were recorded, they were nomal in only thirteen. 9. Injury to the ciliospinal tract in its intracranial course is a more probable cause of the Hutchinson pupil and the other pupillary changes than injury to the third nerve or to the cortex, though no single lesion explains all cases. 10. The reflexes may be lessened or lost in fracture of the base, as in any case of violent jarring of the brain. On the other hand, they may be increased even to spasticity, probably through direct pressure on the pyramidal tract as by hemorrhage. It is probable that the initial result of the impact in all cases is a tendency towards lessening or loss of the reflexes. 11. Profuse and persistent bleeding from the ear does not suggest middle meningeal hemorrhage. No middle meningeal hemorrhage was found in the cases of profuse and persistent bleeding, and, conversely, hemorrhage from this artery occurred eight times without, and once with only slight, bleeding from the ear.

Noyes (New York.)

Polyneuritis. L. Harrison Mettler (Medicine, July, 1904).

Author states, as a result of the increasing number of observations revealing central as well as peripheral lesions in multiple neuritis, the opinion is fast gaining ground that in the toxic and infectious case, at least, the peripheral degeneration is dependent upon the central damage, organic or functional, of the nutritive cell bodies of the respective neurones. In other words, the peripheral changes are secondary in very many of the cases, at least, and are poliomyelitic in origin. Many conservative pathologists still believe, however, that there is a direct effect of a destructive character exerted upon the peripheral nerves by the toxin simultaneously with the effect of the same poison upon the central elements.

J. E. Clark (New York.)

The Reflexes in Alcoholism (Deut. Med. Woch. No. 2, 1904).

Gudden has described a slow reaction of the pupil in acute alcoholism. Kutner has noted the condition of loss of muscular tone and tendon reflexes in the same condition. In cases under his observation transitory disturbances of consciousness and fixed ideas were noted. Typical epileptic attacks and hemicrania were seen. In all these cases the reaction appeared after a relatively small amount of alcohol had been taken, and sometimes when they had been abstemious for some time. Sometimes the knee-jerks were increased, indicating central irritation. In all these cases of intoxication a marked increase of the passive mobility of the limbs, hypotonia and feebleness of tendon reflexes were noted. Skin, plantar and abdominal reflexes were variable. The corneal and conjunctival reflex were constant.

Noyes (New York.)

Overwork and Myasthenia Grave. Pel (Berlin klin. Woch. No. 25).

The author observes that myasthenia, with or without bulbar paralysis, is found to be related to the morbid phenomena of mysteria, with the cardinal symptoms of grave neurasthenia, or with the cerebral or spinal symptoms of multiple sclerosis, and with syphitis of the nervous system. He publishes a case where neurasthenic phenomena predominated. Concerning atrophy of the tongue, he states that muscular atrophy does not of necessity belong to grave myasthenia, and the lack of reaction of degeneration is characteristic. Atrophy of the muscles of the neck may occur. Oppenheim suggests the term of myasthenia pseudo-paralytica. Overwork of certain muscles is the chief cause of the disease.

Noyes (New York)

A SPORADIC CASE OF CEREBRO-SPINAL MENINGITIS, WITH A DESCRIPTION OF THE PATHOLOGICAL AND BACTERIOLOGICAL FINDINGS. Robert Reuling (Maryland Medical Journal, June, 1904).

Autopsy.—Negro, age forty-three; post-mortem thirty-six hours after death. Microscopic examination, cord tissue hardened in ten per cent. formalin. Eosin, hemotoxylin stains. Marchi, osmic-acid stains. Dorsal cord, intense purulent infiltration of pia. Pus cells mostly polymorphonuclear leucocytes, in various stages of degeneration; few lymphocytes; few "Mast-zellen." Eosinophilic leucocytes not found. No fibrin formation. No fibrinoblasts apparent. Cord substance shows only moderate infiltration by pus cells. Occasional pus cell found in central canal. Numerous round bodies, simulating cells, very abundant where meningeal inflammation most intense. These bodies apparently derived from degeneration of myelin sheaths. Marchi staining exhibits profuse myelin degeneration throughout white substance, especially in nerve fibers at periphery of cord. Bacteriological examination. Cultures form pus at base of brain and subdorsal space in cord. A pure culture of the diplococcus of Jäger-Weichselbaum, obtained on agar-agar, also blood serum.

J. E. CLARK (New York.)

CEREBRAL GYRI IN PARESIS. L. BOWMAN (Psychiatrische en Neurologische Bladen, Vol. 9, 1904, No. 4, July, August).

The relative weight of the cerebral gyri in twenty-five cases of general paresis. Winkler has shown that with sufficient exercise one can divide the cerebrum in a number of comparable parts, viz.: after careful fixation and hardening in the same fluids after the same method. The differences in weight of the two hemispheres are of little importance. The relation between the weight of the pallium and the basal ganglia in normal cases and in general paresis are very different. The relation is in normal cases 12, 2:1, and in G. P. 10, 26:1. The frontal lobe is in G. P. very diminished in weight. In this disease there is a marked difference in the weight of right and left hemispheres; after six and more years no such marked difference is found.

MUSKENS (Hague.)

FIBRO SARCOMA OF THE NERVOUS SYSTEM. Hulst (Psychiatrische en Neurologische Bladen (Vol. 8, 1904, No. 3, May, June).

After having given a review of the current literature of multiple neurofibroma in the nervous system, the author relates two cases of his own observation. The first one involves the central apparatus, the second one the peripheral nervous system. As well the clinical as the anatomical aspect of his subject is explicitly discussed. As an accidental complication in the second case syringomeylia was found. Of this last disease the clinical symptoms were nearly entirely wanting. The conclusions to which these cases lead the author are: 1. The affection, known under the name of multiple neurofibroma, neuro-fibromatose generalisé, neuro-fibromatosis, so-called tumors of the acoustic nerve, tumors of the "kleinhirnbrückenwinkel," etc., belong all together to one process, that is, fibrosarcomatosis or fibro-sarcoglio-matosis of the nervous system. 2. This affection belongs to the exquisitely degenerative diseases, and are accompanied by different stigmata, which tend to show the defective and incomplete development of the individual. These stigmata are as well of a somatic as of a psychical nature. 3. The appearance of these symptoms do not depend upon age. They may commence to appear as well in the early as in the advanced age. 4. In the majority of cases there is a distinct progressive character. 5. The entire nervous system, without any exception, is involved in the disease.

MUSKENS (Hague.)

Book Reviews

EPILEPSY AND ITS TREATMENT. By WILLIAM P. SPRATLING, M.D., Medical Superintendent of Craig Colony for Epileptics. W. B. Saunders & Co., Philadelphia, New York and London.

From a rich experience of many years as Superintendent at Craig Colony for Epileptics, Dr. Spratling has given us a really splendid book. It certainly occupies a position in the front rank with Gowers, Féré or Binswanger. From the practical standpoint we consider it the superior of all.

The feature that stamps this book as one well worth having is its evident first hand knowledge. While bibliographic research is not at all neglected, and while the opinions of other authors are freely quoted, it is evident throughout the book that Dr. Spratling is dealing with epileptics and not with books about epilepsy.

The chapters on treatment are the fullest, and, to our minds, the most useful that we have seen. A little too much space has been given to the utterly rubbishy hypothesis concerning eye-strain and epilepsy, but inasmuch as this and many other similar half-baked notions are hard to down in the face of their persistent advertising, a definite statement concerning them in a work of this kind may be worth while. Similar attention, perhaps for the same reason, has been given to the many quack remedies for epilepsy. We commend this work most heartily. JELLIFFE.

LA MIMICA DEL PENSIERO, STUDI E RICERCHE. DR. SANTE DE SANCTIS, DELLA R. UNIVERSITA DI ROMA. Remo Sandron. Milan, Palermo, Napoli.

Following the studies of Darwin, Hughes and others, de Sanctis makes an application of these inquiries into the relations of expression to thought and the emotions.

He enquires how far can a study of facial expression, of bodily movement, etc., enable one to judge of many of the psychological processes in the absence of voluntary aid on the part of the sick or the insane.

He further focusses his study on the process of attention, and seeks to determine quantitative variations in this fundamental psychological process by close observations on the quality of respiration, the frequency and rhythm, circulatory phenomena, reflexes, pupillary changes and other psychophysiological criteria.

The small volume is one that contains many suggestions, and is well worth while. JELLIFFE.

THE DOCTOR'S RECREATION SERIES. Edited by CHARLES WELLS MOULTON. Volume 1, The Doctor's Leisure Hour. Arranged by Porter Davies, M.D. Volume 2, The Doctor's Red Lamp. Selected by Charles Wells Moulton. The Saalfield Publishing Co., Chicago, Akron, Ohio, and New York.

Of these two volumes the first, as its title indicates, is in the lighter vein, being a collection of sketches, verses and jests, bearing more or less directly upon the medical profession. Some of them are of classic reputation, like the story of the blistering of O'Grady, from *Hany Andy*, and others can trace their descent no farther than the "funny column" of some recent newspaper. Occasionally a touch of tragedy creeps in, but for the most part the contents of the various "departments," classified under such headings as "The Prescription," "The Desperate Case," "General Practice" and the like, is calculated to elicit the laugh which the compiler of this volume rightly considers a valuable remedy.

The second volume avows itself "a book of short stories concerning the doctor's daily life." Collected from various sources, the tales range from grave to gay, presenting some of the many tragic, curious, pathetic or humorous episodes in which the doctor's career abounds.

Each volume has four full page illustrations, and appears in attractive binding and readable print. They make very desirable waiting-room books.

POPE.

MANUEL POUR L'ETUDE DES MALADIES DU SYSTÈME NERVEUX. Par le DR. MAURICE DE FLEURY. Felix Alcan, Paris. 25 francs.

This is a volume of 1,000 pages, devoted to the exposition of nervous diseases. Dr. Fleury has published in times past two particularly interesting volumes, on troubles of the mind and on neurasthenia, and he brings to this special study the same qualities which have made his former volumes so widely recognized.

Fleury's work has always shown singular lucidity, combined with a distinct power of description, and these attributes contribute to make the opening chapters, devoted to the minutæ of case examination, interesting as well as instructive. At least 110 pages are devoted to this aspect. Considerable attention is devoted in a second part of the volume to the histology and anatomy of the nervous system. Here many pchematic illustrations are well introduced, and the fundamental facts of nerve histology, anatomy and physiology well presented. The more detailed description of nervous disorders then follows. The system diseases of the spinal cord first being taken up. Then follow in much the classical order other affections of the nervous system.

The work may be cordially recommended. The author shows himself thoroughly en rapport with his subject, and the mode of presentation is clear and convincing. Considerable and detailed attention is given to the treatment of the various diseases under consideration. GOODALE.

A MANUAL ON PSYCHOLOGY. By G. F. STOUT, M.A., Camb., M.A. Oxon., LL. D. Aberdeen, Wilde Reader in Mental Philosophy in the University of Oxford; Examiner at London University, etc. Hinds & Noble, New York.

This book, judged in the light of its avowed purpose, is a distinct success. It is aimed at the needs of the college student, the average undergraduate, and the author in his preface declares the intention of "avoiding sketchiness" and the desire to "clothe in living flesh and blood" his introduction to psychology.

His exposition is made from a genetic point of view, and the titles of the main divisions of the book, "General Analysis," "Sensation," "Perception," "Ideational and Conceptual Process," will give a general idea of it structure. A point to be commended is the careful clearing of the ground in the preliminary chapters on "Scope of Psychology," "Data and Methods," and "Body and Mind." Definition of terms and distinct limiting of subject, always an invaluable factor in exposition, is never more so especially in a book of this character, and the author has been at obvious pains to select such incidents as will, to quote again from the preface, "give the beginner a real interest in the subject and a real power of dealing with it, even when formulas fail him." The intention, in short, is to make psychology and "psychological thinking" a thing of daily life, not an occult and abstruse matter, veiled in unexplained and mysterious phrases. That this intention has been admirably carried out a very cursory examination of the book will reveal.

A. S. POPE (New York).

THE PHYSICIAN'S VISITING LIST, 1905-1906. Blakiston's Son & Co., Philadelphia.

Any busy physician will find this little book a most attractive example of *multum in parvo,* enabling him to summarize, with the least possible time and effort, his professional accounts and records. It is tastefully and strongly bound in black leather, is furnished with a pocket and pencil, and is of convenient size for carrying in one's pocket. G. G.

TRATTATO DELLE MALATTIE MENTALI. Del PROF. E. TANZI, Direttor della Clinicà delle malattie mentali e nervose nel R. Institute di Superior di Firenze. Societa Editrice Libraria. Milano.

Preliminary Considerations, The Seat of the Psychical Processes, The Causes of Mental Disease, The Anat-pathological Substratum of Mental Disease, Sensation, Ideation, Memory, The Sentiments or Feelings, Movements and External Reactions, these are the titles of the introductory chapters in this most recent and extensive of Italian works on psychiatry.

They are in many respects most individualistic, fresh and instructive. They present the general symptomatology of insanities in the new light of the most recent clinical and psychological research. Being less polemical and more dogmatic than many modern German treatises these chapters have a distinct pedagogic value.

Chapter IX., p. 239, takes up the classification of the insanities. A short historical résumé is inevitable, and a brief criticism of the various systems. The relation of Kraepelin's system to Morsellis' is interestingly brought out, and the author outlines as his own scheme a few broad groups as follows: Poisons, with pellagra, alcohol, drugs, etc.; Toxic Infections and Autoinfections, with amentia, uremic and thyroid psychoses and general paralysis; Encephalopathies, acquired idiocy, tumors, hemorrhages, etc.; Affective Psychoses, retaining mania, melancholia and circular insanity; Constitutional Neuropsychoses, neurasthenia, epilepsy and hysteria; Dementia Præcox, with Kraepelin's classification; Degenerative anomalies, and ontology are inevitably suggested to the student. In this case, while recognizing this inevitable suggestion, the author, by skillful partition of the matter treated, narrows his field to manageable limits. Throughout the book he keeps in mind the peculiar requirements of his special audience, and guards at every point against puzzling vagueness or misleading speculation. Certain fallacious theories which have gained historical significance he does touch upon, and wisely, since the student is likely to encounter them in collateral reading; but otherwise he confines himself to a presentation of the accepted facts of psychology.

Following are the descriptions of the various clinical groups as outlined in the scheme of classification. Thus far only the fascicles on the intoxications have appeared. The work thus far is one of much interest, but we reserve further analysis until the work is completed.

JELLIFFE.

LEHRBUCH DER NERVENKRANKHEITEN FÜR AERZTE UND STUDIERENDE. Von PROF. DR. H. OPPENHEIM, in Berlin. Fourth Edition. S. Karger, Berlin.

Oppenheim's new edition, the fourth, appears in the original German this time in two volumes, serially paginated, making a single volume of 1,500 pages.

Three years only have elapsed since the appearance of the third edition, and yet so many have been the newer investigations bearing on the nervous system that the record of these years has necessitated a very marked revision of his previous classical volume. This rewriting has resulted in a new work of many additional pages and many interpretations, varying in some degrees from those put forth in the third edition.

Inasmuch as the author has amplified his bibliographical citations very considerably he has felt it incumbent on him to explain that it still remains his intention to keep the work within the bounds of a text-book, and thus has been compelled not to unduly enlarge the citations of names of the authors of numerous researches, but feeling the criticisms from numerous sources—in some of which we ourselves have joined—that he has not given as careful attention to French literature as he might, he expressly acknowledges his lapses in this particular, and has taken particular pains in this edition to give full credit to these French workers. Much to the betterment of his volume and to its catholicity, in our opinion.

The work has grown so large that it is impossible to take up its parts and seriously discuss the author's present conclusions. In the main they are those he has held for some time, hence we are relieved of the necessity of such an analysis. We note that all the newer diagnostic features are included, such as, for instance, cytodiagnosis in tabes and general brain paresis, but such would be expected from an author of Oppenheim's reputation. We congratulate the author on his new edition; particularly on the enlargement of his Teutonic field and the inclusion of some of the work of his gifted Gallic confreres. To all who do not possess an Oppenheim we believe it to be a necessity as a working basis of the most fundamental facts of neurology put in logical and orderly arrangement.

JELLIFFE.

ESSENTIALS OF NERVOUS DISEASES AND INSANITY, THEIR SYMPTOMS AND TREATMENT. By JOHN C. SHAW, M.D., late Clinical Professor of Diseases of the Mind and Nervous System, Long Island College Hospital Medical School. Fourth Edition, Thoroughly Revised by SMITH ELY JELLIFFE, M.D., Ph.D., Clinical Assistant Columbia University, Department of Neurology, Visiting Neurologist, City Hospital, New York. Illustrated. Philadelphia, New York, London. W. B. Saunders & Co., 1904.

The original author of this little book did not claim for it the purpose of taking the place of larger and more complete works, but only asked that it be considered as a primer for advanced students. Many medical practitioners, however, who have found the subject of nervous and mental diseases an ever-recurring puzzle, to which the larger text books, because of the fact that they required so much time, did not furnish a ready key, will discover in this little book most of what they needed to guide them through the mazes of these difficult subjects. Even the suggestions for treatment are clear and definite, always helpful and always suggestive. In its present form, in its fourth edition, it has been brought well down to date, and deserves to be even more popular than it was before. In the department of mental diseases particularly, the improvement in the volume is marked, and the most recent contributions to this branch of medicine have been taken into consideration. WALSH.

NIETZSCHE. Von P. J. MÖBIUS. Ausgewählte Werke, von P. J. MÖBIUS. Vol. V. Johann Ambrosius Barth, Leipzig.

His was certainly not an attractive face, Nietzsche's, if we are to judge from the title page half-tone of a bust by Klingers, made from a death mask, but one of commanding attention.

Möbius has given us another edition of his life of Nietzsche, in which he has been able to add somewhat to his earlier description, make a few corrections and incorporated a few notes of personal interest. The extraordinary brilliancy of Nietzsche challenged the attention not of the German world alone but of the entire globe, and we are indebted to Möbius for a sympathetic outline, more particularly of the great mental accident that befell him and of his gradual mental deterioration, rather than for any philosophical critique of his system, his writings or his general theories.

There have been other medical biographers, some paragraphers, many superficial faddists, who have never understood, or have not cared to understand, the medical aspects of Nietzsche's case, but in Möbius' account we believe there is a clear consecutive and comprehensible outline of the rise, the development and the fruition of the disease, general paresis, occurring in a man of unusual genius, contributing in some part to his eccentricities, if not to his paradoxical hyperboles, and finally killing him. Nietzsche, to begin with, was not an entirely well man—no one expects a true genius to be absolutely well—the stress of great mental activities precludes the minor cautions of life that ensure average good health. The constitutional migraine from which Nietzsche suffered, and from which, if dispensary and private records are worth much, practically everybody in urban life suffers, was more than the average indisposition of the many—it was a severe trial, and it with other weaknesses gave an impression of a distinctly asymetrical man-type in Nietzsche. That the general paresis was a pure accident we readily admit. If the teachings of the believers in accidentally acquired syphilis are true, the greater wonder is that more of the great minds of the world do not give out under the added stress of a post-luetic arteriosclerosis. For the small preachers alone the moral aspects of the question are worthy of consideration. It is not certain just when the disease commenced to show itself in Nietzsche's case. It is known that as early as 1888, while at Turin, he had a seizure of a peculiar nature, presumably apoplectiform, but it is not improbable Möbius thinks that the signs of paresis were present before this, but it is a difficult matter to trace them in his writings. So far as heredity is concerned Nietzsche's father died young, thirty-three, probably of a brain tumor—there was some insanity in the mother's family, but Möbius would not dwell upon these facts as anything more than of passing moment. In "thus spake Zarathrustra." Möbius would see the more distinct beginnings of mental breakdown. From this on, 1886, to his death in 1900, the progress was slow but distinct. The disease certainly persisted eleven years in Nietzsche's case, if not more. The physical stigmata were distinctly visible when he was treated at the Baale clinic in 1890, ten years before his death. We would refer our readers to the original of this highly instructive biographical sketch, and congratulate the author on his sympathetic and thorough treatment of a difficult matter. JELLIFFE.

MENTAL DEFECTIVES, THEIR HISTORY, TREATMENT AND TRAINING. By MARTIN W. BARR, M.D. Philadelphia, P. Blakiston's Son & Co., 1904. P. 337. Price, $4.00 net.

The author of this work, who is chief physician of the Pennsylvania Training School for Feeble-minded Children, at Elwyn, states in his Foreword that it is "The result of an experience of almost twenty years in carrying forward principles of treatment and of training, tested, proven and defined—it addresses itself primarily to anxious parents and to earnest teachers, rather than to the scientist." For the purposes here set forth the work is most admirably adapted. It is quite free from anything that savors of pedantry, setting forth the various aspects of the subject in a clear and comprehensive way and in simple language easy to understand. On the other hand, the results of twenty years of work among the feeble minded with various methods of treatment, manual training, etc., and the careful statistical studies of hundreds of cases bearing on questions of etiology, cannot fail to interest the expert. Following a most interesting chapter, dealing with the history of the subject in hand, the author takes up the subject of classification. The classification suggested is intended to be primarily practical, and is based solely upon the educational possibilities of the different degrees of defect, which are broadly designated by the terms, "idiot," "idio-imbecile," "moral imbecile," "imbecile," and "backward" or "mentally feeble." Each of these classes is then further subdivided in accordance with their capacity for training. The subject of etiology is discussed at length. The chapter is one of the most important in the book. Great stress is laid upon abnormal conditions of the mother during gestation, especially such conditions as result in exhausted vitality. Now that the great war in the East is engaging so much attention it is interesting to speculate on some of its remote results in the light of the information furnished by a French military surgeon in reference to the siege of Landau in 1793, "of the ninety-two children born within a few months, of mothers exposed to the terrors of cannonading and the blowing up of the arsenal—sixteen died at birth, eight were idiotic, and died before they attained the age of five years; thirty-three, more or less defective, died within ten months, and two were born with fractured limbs." The chapter devoted to training is well written and readable. Educational methods should not be begun before the seventh year, and the period of receptivity closes on the average at the sixteenth. Special stress is laid on the utter hopelessness of cure. All that can be done is to awaken dormant faculties, making the most of what exists; new powers cannot be created by education—"that which the cradle rocks the spade will cover." In the chapter on echolalia a most interesting case is reported, with the general conclusion that the symptoms indicate that echolalia is in reality a transcortical motor aphasia. The work is profusely illustrated by photographs, illustrating the different types of feeble mindedness, and accompanying each is a short clinical history. Many interesting cases are cited, and the last chapter is a collection of instructive writings and anecdotes. WHITE.

News and Notes

APPOINTMENT OF DR. PRITCHARD.—Dr. W. B. Pritchard has been appointed Professor of Neurology at the Polyclinic Hospital and Medical School, to occupy the chair recently vacated by the resignation of Dr. B. Sachs.

NEUROLOGY AT PAN-AMERICAN CONGRESS.—An invitation is given to all members of the profession interested in neurologic and psychologic medicine to attend the neurologic section of this congress, January 2-6, 1905, at Panama, to present papers or other communications in the line of the section's work, by Dr. C. H. Hughes, 3857 Olive Street, St. Louis, Secretary of the Section of Nervous and Mental Diseases.

THE NEW OFFICERS OF THE NATIONAL EPILEPSY ASSOCIATION.—At the fourth annual meeting of the National Association for the Study of Epilepsy, held in Boston, November 22, the following officers were elected for the coming year: President, Dr. W. P. Spratling, of Sonyea, N. Y.; First Vice-President, the Hon. W. P. Letchworth, LL.D., of Portage, N. Y.; Second Vice-President, Dr. Max Mailhouse, of New Haven, Conn.; Secretary and Treasurer, Dr. Everett Flood, of Palmer, Mass.; Chairman of the Executive Committee, Dr. William N. Bullard, of Boston. The next meeting will be held in New York in November, 1905.

NEW PSYCHIATRIC CLINIC AT MUNICH.—The new clinic was formally inaugurated by Professor Kraepelin, November 7. It is said to be the most completely equipped and convenient institution of the kind yet erected. It has 100 beds and a number of innovations designed by the late Professor Bumm.

CLARENCE L. HERRICK.—The death of Prof. Clarence L. Herrick took place September 15 in New Mexico. We quote an appreciation of him from *Science:* "In him neurology and geology alike have lost a brilliant investigator and a teacher of rare power.

"His scientific work began in the high school. During his college course at the University of Minnesota, where he graduated in 1880, he was employed on the Natural History Survey of the State, and for five years following he was actively connected with this work, completing a large quarto on the mammals of Minnesota in 1885. From 1884 to 1889, and again from 1892 to 1894 he held a professorship at Denison University. Here he continued his zoological studies, but in connection with his class work became interested in the Waverly limestones and shales of Ohio. He devoted himself to the study of these for several years with charactristic intensity, publishing most of his results and those of his students in the *Bulletin of the Scientific Laboratories of Denison University,* which he founded in 1885. From the first his teaching was extraordinarily successful, particularly in kindling enthusiasm and love of research. This was due partly to his attractive personality, partly to his fearless originality, but chiefly to his philosophic insight and his ability to open up his deepest thinking even to elementary pupils. And so a very large proportion of his students have themselves achieved success as original workers in science.

"During his last ten years, spent in New Mexico on account of the breakdown in health, which forced him to leave Ohio, he resumed his geological studies, publishing several important articles on the geology of that territory.

"From 1889 to 1891 he was professor of zoology in the University of Cincinnati. Here his geological labors were interrupted, and he entered with great energy into a series of neurological investigations which he had long before planned to undertake. He founded the *Journal of Comparative Neurology and Psychology*, which (now under the editorship of his brother, C. Judson Herrick) has made a permanent and important place for itself. Beginning his neurological work upon the brain of rodents, he accumulated a large mass of data which he found almost incapable of correlation. Believing that the key could be found only in lower primitive types he began to examine a large number of such in a very thorough manner. His results were published rapidly and with little attempt at correlation. These papers were illustrated by large numbers of beautiful plates, which his rapid and skillful use of the pencil made possible. His plan was to secure a large amount of accurate data while his eyesight was still perfect, and later review the whole field of vertebrate neurology, using his own observations as a nucleus around which to build a unified system by further research at critical points. In 1892, after some months of study abroad, he returned to Denison and continued his neurological work with great energy, until in December, 1893, failing health compelled him to go to New Mexico. He soon recovered sufficiently to resume work, but local conditions were such that his attention was again directed mainly to geological problems. The work of correlating his neurological studies was left somewhat incomplete, but it is probable that this may be accomplished through the labors of his brain-children.

"For four years he did a useful work as President of the University of New Mexico, here as everywhere stimulating young men to undertake research by the influence of his own example. In these later years we see the successful struggle of a noble soul dominated by a great purpose over the discouragements of physical weakness and suffering."

A. D. COLE.

DR. JOHN A. CALDWELL has resigned his position as Resident Physician to the Cincinnati Sanitarium, to enter private practice in Cincinnati.

DR. CHARLES B. ROGERS, formerly of Massillon (Ohio) State Hospital, and recently Assistant Physician to "Fair Oaks" Sanitarium, has been appointed Resident Physician to the Cincinnati Sanitarium.

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VOLUME 32 FEBRUARY 1905 NUMBER 2

THE
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Original Articles

THE COURSE OF THE SENSORY FIBERS IN THE SPINAL CORD AND SOME POINTS IN SPINAL LOCALIZATION BASED ON A CASE OF SECTION OF THE CORD.*

By MORTON PRINCE, M.D.,
of Boston.
PROFESSOR OF NERVOUS DISEASES, TUFTS COLLEGE MEDICAL SCHOOL.

The afferent fibers in the cord pass upwards in the dorsal and antero-lateral columns. Which tracts convey sensory impulses, and whether there is a crossing in the cord has not yet been proved.

Function of the Posterior Columns.—The function of the long posterior columns has never been definitely made out, though it has been well established that the path is largely made up of long fibers which coming from the posterior roots of the same side ascend directly to the dorsal column nuclei in the bulb. From here the next neurone in the chain crosses the median fillet to the opposite side to ascend by paths which are still the subject of dispute to the cerebral cortex.

Experimental Evidence.—Aside from the contradictory results of experimentation in animals, this kind of evidence is unsatisfactory inasmuch as there is reason to believe that the course of the sensory tracts in animals differs from that in man.

Schiff,[1] from his experiments, maintained that after transection of all the columns except the dorsal, signs of sensation could still be evoked from the parts below the section. Herzen[2] and others confirmed this view that these columns were especially for tactile sense. Osawa,[3] on the contrary, in similar experiments found sensation entirely abolished. Indeed severence of the posterior columns has been performed by different experimenters many times[4] at different levels without perceptible impairment of sensation. Borchert[5] gives a careful résumé of the results of posterior and lateral tract section in 11 dogs and 2 foxes. In no case where the posterior columns alone were cut was there any disturbance of tactile perception.

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* Read at the meeting of the American Neurological Association, June 5, 6 and 7, 1902.

The impression is growing from recent experiments[6] that the posterior columns are largely for the conduction of muscular rather than tactile sense, and there is much in favor of the view adopted by Hering,[7] that they serve to conduct centripetal impulses which, arising in the apparatus of movement, regulate by unconscious reactions the movements of the skeletal musculature. Yet Meyer's case seems to show that they are capable of conducting tactile sensation.

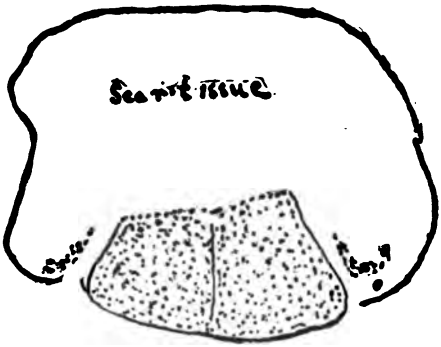

Fig. 1.—Cut from Meyer's article.

The lack of pathological data in most cases of incised wounds in the cord, and the absence of sharply circumscribed lesions in the other cases that have come to autopsy have made physiological study from this source difficult. Meyer's[8] case is important in that the lesion ("Myelitic destruction") was sharply defined though of slow development. There was destruction of the entire cross-section from the 4th to 6th thoracic segment, with the exception of the dorsal two-thirds of the posterior columns and a few pyramidal fibers which were intact. (Fig. 1.) "Tactile sensibility is normal, also localization," the report says, but analgesia and thermanesthesia were present below the 6th rib. It is to be regretted that the method of testing tactile sensibility is not given, particularly as the patient was insane and presented marked defect of memory suggestive of Kortchakoff's psychosis (anterograde amnesia). Details of the microscopical appearances and the technique employed are not given. The possibility that loss of function was not as great as the anatomical appearances indicated (as so frequently is the case) must be considered and might explain the loss of tactile sensation, otherwise it would seem that tactile sense must have been transmitted by the posterior columns in this case, though pain and temperature impressions failed to be transmitted. If this is the rule for tactile impressions it is hard to explain why many cases of tabes with extensive degeneration of the posterior columns should have little or no disturbance of tactile sensation unless there are other paths of sensation. Further the supposition that the posterior columns are the sole or chief conductors of sensibility is incompatible with the clinical condition known as Brown-Séquard paralysis where the crossed sensory paralysis below the lesion indicates a crossing in the cord. The posterior tracts cross only above in the bulb. Another possible interpretation of Meyer's case is that the development of the lesion, being gradual, the posterior columns took on functions that ordinarily are performed by the antero-lateral columns. That is to say, that there are two paths for sensation, the chief being the antero-lateral columns. In slow developing lesions of these, the posterior columns may take their functions. This case of Meyer's is the direct complement of my case in which both the posterior columns were cut without loss of tactile sense on one side.

Function of the Lateral Columns.—Tactile Sense.—There is experimental evidence strongly suggesting that at least one of the paths of conduction of tactile sense is in the lateral part of the cord, though this evidence is conflicting as to whether this path is crossed or not. Borchert,[5] after section of the posterior and lateral columns on the right side of a thoracic cord, and the posterior alone on the left, found tactile sense lost below on the right but intact on the left. A similar experiment in a lumbar cord gave a corresponding result. These experiments were both on dogs, and it will be noticed that the anesthesia ***corresponded to the side of the lesion.*** This observation agrees with the experiments of Mott[9] and Schäfer[10] on monkeys. Mott concluded that, in these animals, while pain and temperature sense are conducted on both sides of the cord, tactile and pressure sense are conducted only along the same side of the cord. Weiss[11] performed hemisection in the dog, and found that sensibility became after a time restored on the side of the lesion, but remained permanently impaired in the *contralateral* limb differing in this respect from Borchert and Mott. Both Ferrier and Turner, too, as a result of hemisection of the cord in monkeys found that tactile sensation passed up the opposite half of the cord. The difficulty met in my case in determining the exact area incised in each half of the cord impressed upon me a possible source of error in physiological experiments, and a possible reason for contradictory results.

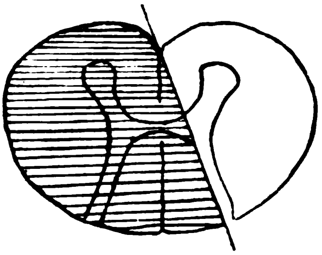

Fig. 2.—Cut from Müller's article.

As to ***pain and temperature,*** the evidence is convincing that their conducting paths are not only crossed but also that, probably after the first synopsis, they run in the lateral columns, though some observations on animals, as those of Mott and others, are interpreted as showing both uncrossed and crossed conduction. Schiff[12] and also Budge[13] erred in concluding from vivisection experiments that pain is conducted in the gray matter, for many experiments show the gray matter incapable of conducting a long distance.[14] They may be right, though, in holding that only those fibers conduct pain that end in the gray matter thus excluding the posterior columns. Brown-Séquard,[15] from clinical studies after wounds of the cord and both Ferrier,[16] and Turner,[17] as a result of the experiments above referred to on monkeys, concluded that *pain* impulses crossed soon after entrance and passed up the opposite half of the cord. Miescher[18] and Woroschiloff[19] also obtained the result that, in the rabbit, these impulses ascend in the lateral columns. Sherrington[20] reached the same result in the dog as indicated by blood pressure impulses. Both Miescher's and Sherrington's experiments indicated that the impulses crossed in the cord. Others[21] claim that the conduction from one side of the body is by both lateral columns in about equal proportion. The value of these experiments has the necessary limitation of observations on dumb animals, and, whatever the reason, this experimental evidence while showing conduction of sensibility in the lateral columns, is conflicting in regard to whether the condition is by the same or the opposite side for tactile sensations, and by only the opposite or both sides for pain and temperature sensations.

Clinical pathology will probably have to settle the questions in dispute. The great difficulty is that of obtaining pure cases of limited lesions. There is some evidence in hand.

Myer's case pointing to tactile conduction by the posterior columns has already been described.

Gowers[22] describes an important case of injury to the cervical cord by a spicula of bone and hemorrhage. As figured in a cut (not very distinct) the injured area was limited to the gray matter and antero-lateral region, bounded behind by the posterior horn and anteriorly by a line not quite as far forward as the anterior roots. It thus involved the triangular region, which in my case seems to contain the paths for tactile sensations. Gowers found entire loss of sensibility to pain (and temperature) *without impairment of tactile sensibility.* (Query: Did the hemorrhage which occasioned the damage in the anterior portion of the triangle actually destroy conduction?) This case is in harmony with that of Meyer, but Gowers erroneously and unaccountably quotes a very important case of Müller as a complement of his own case, and as corroborating the view that the tactile sensations are conveyed by the posterior columns. The case was one of knife stab of the dorsal cord. The *whole of the left half of the cord* was severed, but only the *posterior columns of the right half* (Fig. 2). Gowers states that tactile sensation was lost on both sides, and pain on the right only.

Examination of the original report[23] shows tactile sensation was not lost but only diminished on the left side; i.e., "Slight [oberflächliche] touch from the fourth intercostal space downward was not perceived." On the other hand the left side was very sensitive (sehr empfindlich) to "more intense stimulation [intensivere Reize] (pressure, percussion, cold)" and the patient declared that "lebhaftes stechen" were felt. Tested with faradic electricity on the day after the injury a trifling [unbedeutender] current was felt on the left side as very painful. On the right side there was absolute anesthesia for touch and pain; intense stimulations, including moderately strong faradic currents, were not felt at all. Four days later it was noted that somewhat stronger pressure upon the muscles of the lower half of the body on the left elicited severe pain. Later still, on the left, "the hyperesthesia abated; less intense electrical currents were perceived, but not so painfully as before."

The symptoms are thus summed up by the author: "On the side of the section; motor paralysis, hyperesthesia for pressure, pain, and the electrical current, contraction of the pupil. On the opposite side; free mobility and complete anesthesia for all sensory impressions." He further affirms that the syndrome was in entire accord with the Brown-Séquard theory, that the crossing of the sensory conducting tracts takes place throughout the whole length of the spinal cord, etc. The Brown-Séquard syndrome which this case is held to exemplify, is stated to be "paralysis on the side of the injury with hyperesthesia for touch, temperature, pain, tactile sense contraction of the corresponding pupil. On the opposite side preservation of movement with complete anesthesia for every form of sensation."

From this report it is plain that tactile sensation was not more than slightly diminished, (what well might have been due to edema of the uncut portion of the cord) and the completely severed posterior column could not have been the path of conduction for the tactile impressions that were conveyed.

This case therefore is contradictory of Gowers' case, and in entire agreement with the clinical findings in my case.

In this connection it is significant that Bechterew and Holzinger,[24] in their experiments on dogs, found that neither section of the posterior columns together with the anterior columns, nor section of the anterior portion of the cord, (that is, anterior column and anterior portion of the lateral columns) caused analgesia; but that analgesia followed only after section of the whole of both lateral columns or after section of the posterior half of the cord provided that the anterior limit of the section was a little in front of the pyramidal tracts. In other words, pain conduction was found to lie in the same triangular region in which the findings in my case show that conduction of tactile impressions occurred.

Dejerine and Thomas[25] in connection with a case of syphilitic hemiparaplegia with crossed anesthesia, conclude that in man the majority of facts seems to favor total or almost total decussation oi the "sensory fibers in the cord." But they admit this conclusion can not be extended to the higher animals; i.e., in the dog and monkey the crossing is certainly incomplete. The ascending degeneration in their case was almost confined to the direct cerebellar and Gowers' tracts on the side of the lesion. The posterior columns were practically normal. The muscle-sense was normal and tactile sense was only slightly impaired in the non-paralyzed leg, but the pain and temperature senses were much enfeebled.

Recently the idea has been growing that there are two possible paths for tactile sensation, one capable of substitution for the other. In this connection Petrén[26] concludes from a clinical study that hemilesions of the cord give only crossed anesthesia, which may be either a disturbance of all the skin senses together or, more commonly, a dissociated type including pain and temperature senses only (the pressure or tactile sense being normal). The great majority of cases of disturbed tactile sense show also paralysis of the opposite limb. Petrén concludes that tactile sense in the cord is conducted over two paths,—one in the uncrossed tract of the posterior column, the other following the tracts of common sensation. These latter cross,—for the lower extremity,—at the 12th dorsal or 1st lumbar segment and gradually work laterally till they lie about at the root of Gowers' tract. These conclusions are in harmony with Meyer's and my case.

Ballati's[27] recent conclusions are similar, and he adds that dissociation of sensibility by medullary compression must arise from incomplete lesion of Gowers' tract.

It has been assumed that a pain tract does not also conduct tactile impulses but, in spite of the extensive work of Herzen[2] and

Goldscheider[28] in this direction, it is hardly proven. Pain may be only a result of an intensification of the impulses that ordinarily would be felt as touch.

To summarize, it is impossible to harmonize all the experimental evidence bearing on the various problems which arise in regard to the transmission of sensibility in the cord, and particularly of tactile sensation. Anatomically one must expect the conduction in the cord to be by either the posterior or antero-lateral tracts, or by both. The case here reported proves that tactile sensation is conducted through the antero-lateral columns, and it is difficult to interpret the findings unless this conduction is crossed. The supposition of two possible paths of tactile conduction would explain the otherwise contradictory cases of Meyer and the author. Moreover, as already stated, the case of Meyer might be further explained on the double path basis by presuming, since the lesion was of slow progress, that the second path could readily take up the lost function, while in sudden onset there would be temporary anesthesia. This idea, too, would agree with the findings of Brown-Séquard[29] where the hemisection of the cord at different levels made him abandon his well-known theory.

Case of Stab Wound of the Cord.—The value of the following observation lies in the fact that the case was one which was practically of the nature of a vivisection by which the cord was partially severed in the cervical region. Although both sides of the body were paralyzed below the seat of the lesion, tactile sensation was destroyed only on one side. It, therefore, would seem, that, first; if we could determine the exact line of incision of the cord on the two sides, we might be able to throw some light on the paths of conduction for the sense of touch; second, as the incision was sharply made between the sixth and seventh segments it would contribute to our knowledge of spinal localization. The vivisection experiment was due to the fact that two Italians got into a brawl and one stabbed the other in the neck with a knife. The point of entrance was one and a half inches behind and one below the mastoid, and the probe showed that the wound went downward and slightly forward and inward to what was apparently the body of the sixth cervical vertebra. As will be presently seen examination of the cord showed this same diagonal direction of the knife. The patient was at once brought to the City Hospital. On entrance, the evening of December 9, about an hour after the affair, it was noted by Dr. Beebe, the house surgeon, and recorded in the records that at this early period "sensation was preserved in the entire left leg, but ***entirely absent in the right leg.***" There was complete paraplegia and bilateral paralysis of certain movements of the arms which will be later described in connection with the localization of movements. The knee-jerks are described as lively. I saw the patient the following morning, perhaps twelve hours after the injury, and a careful examination then showed that this same hemianesthesia existed on the right side, but the patient could

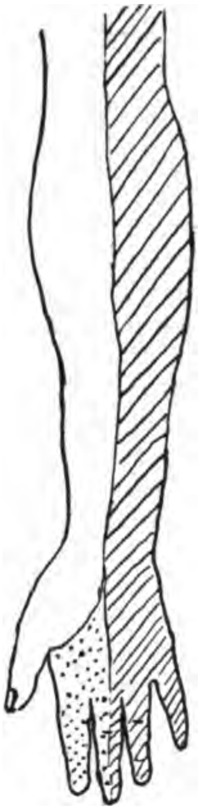

Fig. 3.—Right hand. Shaded area, absolute anesthesia. Dotted area, blunted sensibility.

feel the slightest touch all over the left side: On the right side the anesthesia was profound, so much so that it was easy to map out the line of demarcation in the hand and arm between the normal and anesthetic skin. This line ran down the middle of the forearm and palm of the hand to the middle finger, which was included in the anesthetic area. The temperature sense was not tested. No note was made in the records of the condition of the pain sense. It can not, therefore, be positively stated as a fact that it was lost. But my *recollection* is clear, that a pin prick was used as a test and was not felt; and second, as, in organic lesions, pain is always lost when the loss of tactile sense is absolute, it is highly improbable that the sense of pain was retained. There was absolute paralysis of both legs and of the hands as will be later described. The knee-jerks at this time had become lost, as were all other reflexes in the paralyzed parts.

The injury of the cord was localized correctly as was afterwards proved, at the height between the sixth and seventh cervical segments.

The patient died about four days after the injury on the evening of December 13. At the autopsy the cervical cord was removed with its inclosing vertebræ. The cord, in situ, and the vertebræ were then cut longitudinally, and the whole hardened in formalin. The cord was thus divided, for purposes of examination, into two lateral halves by an incision which passed very sharply antero-posteriorly along the anterior fissure and posterior septum close to or through the central canal. It could be then seen that the stab had made an incision of the cord, which, roughly speaking, severed the posterior half. On the posterior surface the knife of the assailant had cut the cord *diagonally* from above, on the right, to below, on the left, approximately between the sixth and seventh segments. On the right side the incision was sufficiently high to cut two (8th and 9th) filaments of the sixth posterior root, leaving intact the last (10th) filament, while on the left side it passed sharply *between* the sixth and seventh roots and on this side extended forward nearly to a point *half way between the anterior and posterior roots.* On the right side the anterior limit was not at this time determined, but on the median surface the anterior limit of the incision in the middle line could be plainly made out. Here the incision, the edges of which were somewhat jagged from necrosis, extended to and through the longitudinal line marking the gray matter and ependyma around the central canal; (Fig. 4) so that, as was later determined by the microscope, the anterior limit of the section in the median line was just through the anterior

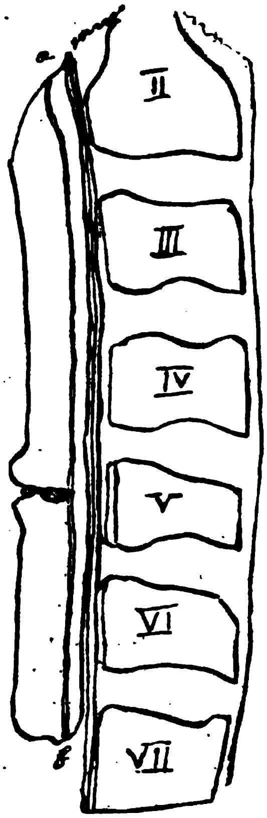

Fig. 4.—Tracing of median surface of left half of spinal cord with vertebræ, made by placing tracing paper over the specimen. The cord has been cut antero-posteriorly along the anterior fissure and posterior septum. The line a b marks the gray matter around central canal. The incision is seen to extend through it.

commissure, and at the periphery on the left side at a point approximately half way between the exit of the anterior and posterior roots. On the right side the anterior limit of the incision was determined by the microscope. The important point was to determine the extreme anterior limit of the incision. For this purpose (after making a series of experiments on another cord in which a similar incision was made), the method was adopted of injecting the incision with a mixture of carmine and mucilage. Under the microscope it was found that the presence of the carmine particles marked the line of incision. Serial sections were then made from a point just above the highest point of incision through the whole of that part of the cord which included the incision. The knife had entered on the right side and, held at a certain angle, had passed downwards, forwards and across the cord. In the transfixion the knife came out farther forward on the left side than its entrance on the right. Thus a greater area of the left half of the cord than of the right was cut, but the *posterior columns were completely severed on both sides.* Approximately the posterior two-thirds of the cord was severed on the left side. As the knife passed slantingly across, it is apparent that at no one level were all the fibers cut, but at each descending level new bundles were included in its swathe.

The peculiar effect of a diagonal cut on a transverse section was to show at each level only a more or less sharply defined slit running from behind forwards, i.e., for example, from the periphery towards the lateral horn on the right, and approximately parallel to the posterior horn. In each level of descent this slit shifted its position from right to left, but kept its same general direction. Approaching the posterior horn, it passed across it, then approaching the median septum, crossed it and finally in a similar way passed over the left lateral columns which were thus extensively severed. This slit, or incision at each succeeding level below was necessarily farther to the left than that in the level above. This will be understood if one pictures to one's self that the effect of a longitudinal cut on each transverse section would be a slit running postero-anteriorly but occupying the same position at every level. In a diagonal cut the slit must shift its position at each level. In some levels in this particular case, particularly on the left of the picture, this was altered by the softening which had taken place in parts of the cut surface converting the slit into a jagged opening, or allowing the specimen to be torn in the cutting. In the microphotographs (Figs. 5 and 6) the carmine granules marking the slit, X, can be seen. Fig. 5 represents the higher level.

In Fig. 6 the slit is approaching the posterior horn. (This peculiar appearance of each transverse section was demonstrated on a model on which a similar cut was made.) This is shown diagrammatically in Fig. 7. The line of incision at each level in the right half of the cord was seen to be full of the injected carmine granules and could be easily recognized; in the left half of the cord the injection was not so successful. The extreme anterior limit of each slit necessarily marked the anterior limit of the section of the cord at each level. By means of an Edinger projection apparatus each transverse section was magnified and transferred to tracing

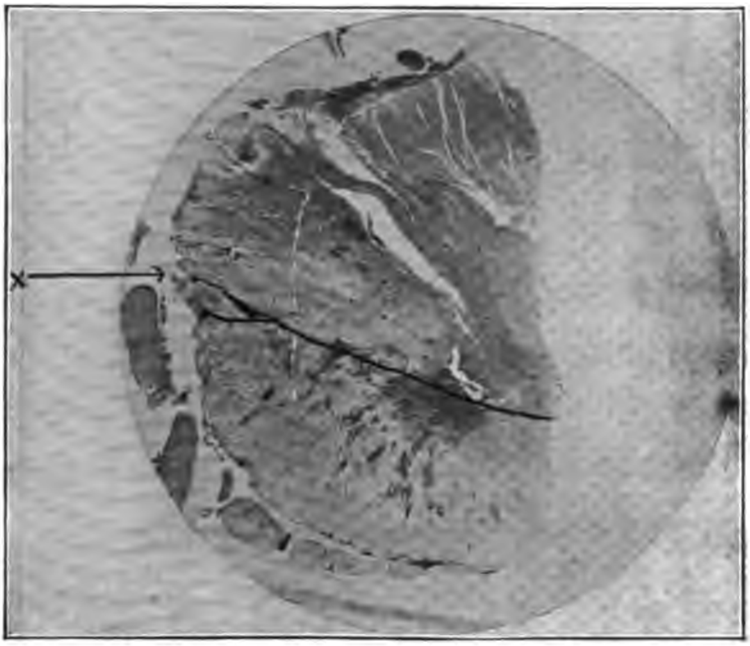

Fig. 5.—Photograph of right half of cord near the upper level of incision. The arrow points to cleft with carmine granules. The black line running from the anterior fissure to the periphery has been drawn on the photograph through the points marking the anterior limits of the incision at each level, plotted on one plane. These points make a straight line. Thus, it will be noticed, the carmine injection at this level terminates in this line. The hemorrhages in the antero-lateral field are conspicuous, but some of the dark spots are blood vessels. The separation of the posterior horn from the white matter is an artifact.

paper. By superimposing each tracing, one upon another, it was easy to plot on a single plane the severed area of the cord. Wherever the carmine adhered every section agreed relatively with one another, and the anterior limits made a straight line from the anterior commissure to the periphery. This plotting, corroborated by the microscope, is shown in Fig. 8, line ab.

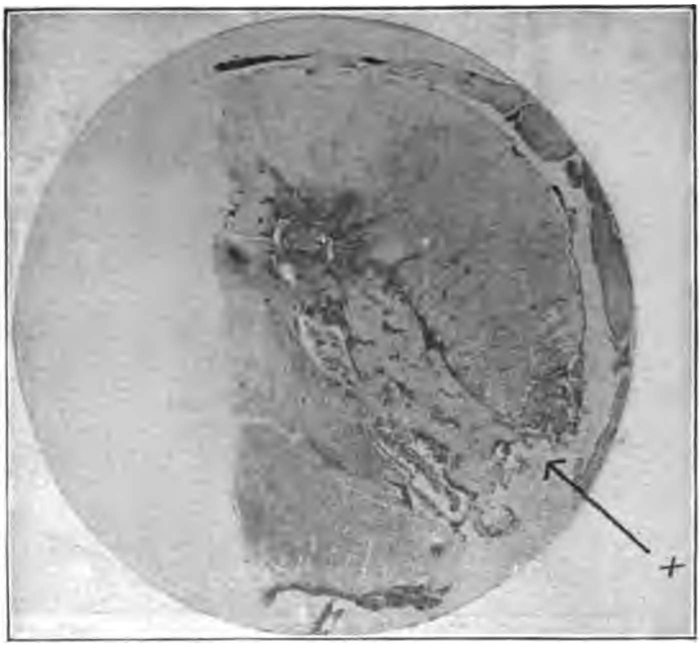

Fig. 6.—Photograph of section at a level a little lower that Fig. 5. Line of incision, X, is approaching the lateral horn, and has extended anteriorly into the anterior horn, which is disorganized by hemorrhage. A second and parallel cleft is seen extending the whole length of the posterior and into the anterior horn. This is probably an artifact, and a continuation of the artifact seen in Fig. 5. Below it disappears. As this area was also cut a little lower down by the incision (X) the findings in any event would not be affected.

In the anterior field, at the higher level (Fig. 5), on the right, there were a number of small hemorrhages scattered and separated by nerve fibres. It might be questioned whether these could have caused the loss of tactile sensation on the same side. If so it would locate the sensory fibers well forward without crossing, but we could not then explain the prevention of sensibility on the left side. Opposed to this interpretation is the fact that the hemorrhages were discrete in islets, while the anesthesia involved the whole half of body. The examination of the specimens themselves does not give the impressions that conduction was extensively interfered with by these hemorrhages.

In the left half of the cord it was difficult to determine the exact anterior limit at and towards the periphery, as the carmine particles did not adhere well, and because in the lowest levels the postero-lateral portion was torn in the preparation owing to softening. Still in a sufficient number of sections the carmine particles adhered to give an anterior line, and at the periphery the free end of the cut membrane gave the terminal point. The various points made a fairly straight line. The anterior limit of the cut area was thus shown by the microscope to be somewhat farther forward than appeared to be the case with the naked eye. It could be determined with positiveness that the section of the left half of the cord extended considerably further forward through the lateral columns than on the right. *This could also be determined macroscopically* so that this fact is indisputable. It will be remembered that macroscopically the incision on the left reached a point approximately half way between the anterior and posterior roots. Further, the microscope showed that numerous hemorrhages had taken place into the left lateral field. These hemorrhages would

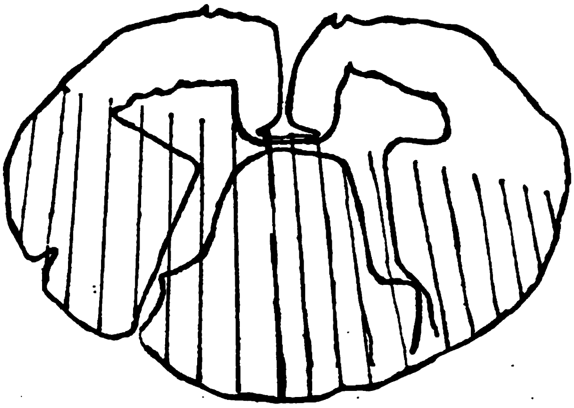

Fig. 7.—Composite diagram showing appearance of incision at each level.

probably have impaired the function of this column even if not cut. The exact anterior boundary on the left side is therefore approximate. (Owing to the short time which elapsed before death the Marchi staining failed to reveal degenerated fibers, and this method could not be utilized.) In the median line the anterior limit of the section was just in front of the anterior commissure.

It is thus determined that the knife entered the cord on the right side at a point that would correspond approximately to the anterior border of the pyramidal tract, then passed inward and forward to the central canal and anterior commissure which it included, swept over to the left, where it followed a line running from the central canal to a point in the periphery still farther forward and somewhat anterior to (or at?) a point half way between the anterior and posterior roots.

Referring to Fig. 8 in which the line of section, ab, has been plotted, it will be seen that the whole of the posterior columns have been divided, and that while on the right side the section passed through the anterior portion of the pyramidal tract without including the antero-lateral field, on the left side a considerable portion of the antero-lateral columns were included. Thus a larger area was cut on the left than on the right.

If we draw an imaginary line, c, on the right, corresponding to the line of incision on the left, we find that we have a triangle, the anterior limb of which will be this imaginary line; the base the periphery of the cord; the apex the anterior commissure, and the posterior limb the line of incision on this side. This triangle is located in the antero-lateral column and represents approximately the additional area cut in the left half of the cord. In includes:—

(a) A portion of Gowers' tract. (b) A portion of the cerebellar tract. (c) A portion of the lateral ground bundle.

Aside from the two first tracts it is well known that centripetal fibers ascend in the lateral ground bundle coming from the gray matter of both the same and opposite sides.

The following conclusions may be drawn from these findings:

First, The whole of the posterior columns and posterior horns were destroyed, and yet tactile sensation was preserved on one side. From this it is positively proved that tactile sensations are conducted by other paths than by the posterior columns, but it cannot be inferred that the posterior columns are not also conduction paths of sensation.

Second, It is probable, thought not certain, that the same conclusions apply to pain impressions (see history).

Third, Inasmuch as the whole of the area destroyed on the right was equally destroyed on the left, if we exclude the scattered hemorrhagic foci on the right, there seems to be no way of interpreting these findings excepting on the supposition that the right hemianesthesia must have been due to the incision of the lateral columns on the *left*. That is, a path for sensibility must cross in the cord; for if the right hemianesthesia were due to the section of the right half of the cord, as the same area was destroyed on the left, left hemianesthesia would also have been present. This is in agreement with the case of incision of the cord reported by Müller.

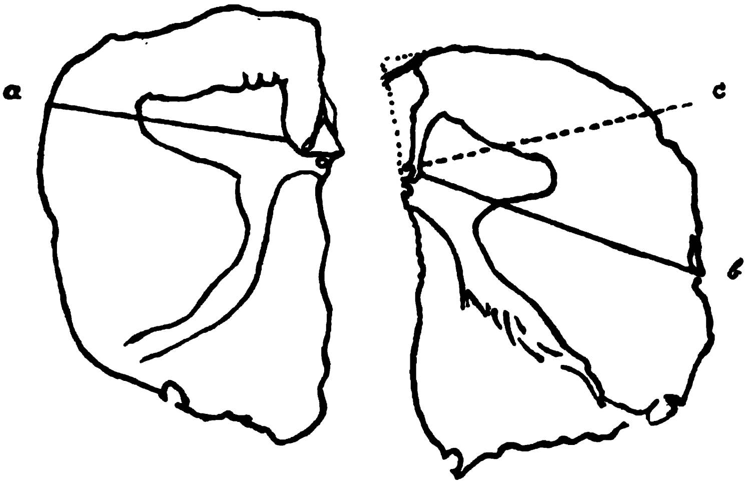

Fig. 8.—Traced and plotted by means of an Edinger projection apparatus from the different sections. The section of the left half of cord is at a lower level than that of the right half; hence the difference in shape of outline of gray matter. The peripheral limit of the imaginary line (C) is taken at the same distance from the anterior fissure as that of the incision (a).

Fourth, A path for the conduction of sensibility must lie anterior to the line of incision on the right and approximately in the area bounded by the above-described triangle in the lateral column; for if this path lies much anterior to this triangle, as it was not cut on the left sensation would have been preserved on the right. (The possibility must be considered that this sensory tract may extend still farther forward, and in this case have been impaired by edema. That the hemianesthesia was found within an hour after the injury indicates that it was not due to secondary inflammation). The only alternative is the location of the tactile conducting path in the anterior field without crossing (seat of the disseminated hemorrhagic foci).

Fifth, There was unilateral priapism, the vascular engorgment being on the left side, that on which the largest area of the cord was divided. The relation of this to the lesion is not clear.

Sixth, As there was absolute paraplegia the uncrossed motor tract could not convey motor impulses of consequence.

Seventh, The anterior horn on the right (the side of the hemianesthesia) was extensively destroyed by hemorrhages. Physiological experiments indicate that sensory conduction is not by way ot the gray matter. This case, however, does not disprove this interpretation. The extensive hemorrhages on the left side were in the white matter and mostly posterior to the line of incision.

Eighth, These findings of crossed sensory paths in the lateral columns are in accord with the clinical Brown-Séquard syndrome, to wit, anesthesia on the side opposite to the lesion in the cord. This syndrome is incompatible with the view that sensory conduction is by the posterior columns; for as the fibers in these columns are known to cross above in the fillet, if they also crossed in the cord there would be a double crossing, and consequently in cerebral disease hemianesthesia would be on the same side as the lesion.

Spinal Localization.—The height of the incision was correctly diagnosed during life, viz., between the 6th and 7th segments. A comparison of the anatomical findings with the clinical manifestations shows some deviations from the usually accepted views of spinal localization. Flexion of the arms was strong and in contrast with the absolute paralysis of extension. The center for the triceps is usually placed in the 6th, 7th and 8th cervical segments. The absolute paralysis of this muscle present in this case in both arms indicates that the center is not above the 7th segment. The wrists could be flexed and extended, though weakly, on both the ulnar and radial side, showing that the center for these movements must extend in part above the 7th segment, at least into the 6th, though Wichmann places the flexors of the wrist entirely below the 6th segment.

Pronation and supination were possible, though impaired; the latter was the stronger.

All movements of the fingers were lost, corresponding to the location usually accepted, though Wichmann extends the centers for the long extensors of the fingers, the flexor longus pollicis, flexor brevis pollicis, and opponens pollicis upward into the 6th segment.

Summing up, the main points for the localization of the lesion between the 6th and 7th cervical segments would be the absolute paralysis of triceps and all movements of the fingers, with normal strength of the deltoid and flexors of the forearm, all other movements being simply weakened.

Sensation.—The line of division between normal and anesthetic skin down the middle of the forearm to the middle finger corresponds very closely with the distribution of the 6th and 7th nerve roots as given by Starr, but differ slightly from that by Kocher, Wichmann and others.

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TWO CASES OF SYPHILITIC DISEASE OF THE CERVICAL SPINE.[1]

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Case I.—J. T. W., aged forty-eight years, Irish-American, a shoe-cutter and form-maker, was seen May 7, 1902. He complained of a stiff neck and very severe pain at a point on the right side of the neck. From this point the pain radiated into the shoulder, arm, forearm and hand. The pain, while never entirely absent, was markedly paroxysmal, and he usually had several very severe paroxysms each day, and many lighter ones. The hardest were at night. The use of the extremity invited the paroxysms to such an extent that he carried the limb much of the time supported by the other hand, and used it as little as possible. He indicated very definitely certain points where he felt the most pain during the paroxysms: viz., the right side and back of the neck, the cap of the shoulder, the anterior and posterior margins of the axilla, the arm through its whole circumference and deep in the bone of the same, the anterior side of the forearm and wrist, and the radial side of the hand; sometimes one, sometimes another of these points being worse. Occasionally the pain would run up to the occipital region in very severe fashion. He always had some pain in the opposite side of his neck, and also fleeting pains occasionally through the left arm. The worse pain was always in the right side of the neck and brachial region, and as the foregoing description shows, it had the character of a cervico-brachial neuralgia. The pain and stiffness had begun together about two months previously and had steadily gotten worse. The patient stated that he had lost forty pounds during that time, was hardly able to sleep at all, had no appetite and was much constipated from the medicine he was taking. He had an emaciated and cachectic appearance. He complained of being very weak and of his legs giving out on exertion. The knee-jerks were quite plus and equal. There was a normal plantar reflex on both sides. On straightening the knees he invariably complained of a pain in the center of the joints and in the patella, but of none in the nerves nor in the hamstrings, although these muscles seemed to be contesting a full extension of the joints. This phenomenon was exactly the same on the two sides. It passed away entirely within a few weeks.

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[1] Read at the meeting of the American Neurological Association, September 15, 16 and 17, 1904.

He complained of paresthesia, especially of a numb feeling about the shoulder and a rather persistent cold spot in front of the wrist, and of a heavy feeling in the whole member. I could determine no objective deviations from normal sensibility, only at times the suspicion of some obtundity. The neck was stiff and the muscles rigid. There was the voluntary fixedness of the head, neck and shoulders due to careful carriage (as some one describes it). The chin was depressed from flexion of the upper segments of the cervical spine. The muscles in the back of the neck had a hard, stiff feel, as though infiltrated, and the skin had a thickened appearance. The region was so painful that satisfactory manipulation could not be made. At this time, with his stiff neck and carriage and emaciated shoulders and pain and plus knee-jerks he presented a pretty fair counterfeit of a case of pachymeningitis cervicalis hypertrophica. Drs. Bliss and Hoge, who saw him, both remarked on this fact.

There was no history of gout or rheumatism. I could not make a positive diagnosis of syphilis, but suspected it for reasons which I will not stop to discuss at this point.

Large and thorough inunctions of mercury to the back of the neck and shoulders were given daily and the iodide of soda was pushed as fast as he could tolerate it. At the end of a week he seemed improved and could get along on fewer pain-powders (acetanilid and phosphate of codeine). At the end of twenty days he was much better. The neck was getting softer and tolerated manipulation so that a deep swelling could be certainly outlined, especially on the right side between the spinous and tranverse processes of the 4th and 5th cervical (possibly 6th) vertebræ. The region was very sensitive. On introducing the finger into the pharynx a lumpy condition of its posterior wall could be distinctly felt, which seemed to be at about the same level as the posterior swelling of the 4th and 5th cervical region. Photographs and X-rays were taken at this time*

At the expiration of thirty days he was still more improved. At about this time he was taking his maximum dose of iodide of 100 grains t.i.d.

A note June 26 (about the fiftieth day of observation) shows that he was not so well after a rapid diminution of doses, the pains getting worse again. The dosage was kept high for another full month.

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* The X-ray picture, although a good one, was of no aid in diagnosis, except in a negative way.

A note on August 31: Has had no pain whatever for two or three weeks. Feels perfectly well. Has regained normal weight, and is at work regularly. The back of the neck is soft and pliable. There is a possible slight tenderness and thickness at the original site in the neck.

January 8, 1903 (eight months after I first saw him), he came with a headache which had all the appearances of a severe syphilitic headache. He had discontinued his visits for three months and had had no medicine for about that length of time. The antisyphilitic treatment was actively resumed and the headache began to yield in a characteristic way. At the end of a month he was feeling quite well again.

At this time I told him he had syphilis. I had before not discussed it with him. He told me that about six years previously he had been treated for the disease (a sore throat and mouth) by a physician of repute in another city, but that he had no later manifestations and therefore had discredited the diagnosis.

Case II.—G. C. S., aged thirty-six years. Canadian, mining-engineer. The day before Thanksgiving Day, 1903, he caught cold at the mines. The day following he noticed a peculiar stiffness or check in the neck so that he could not turn his head freely. That night he was awakened by a severe pain in the left side of his neck. From this time he had more or less pain and stiffness in the neck every day, and the pain was always worse at night. He continued irregularly at his duties until the last Saturday in January, 1904 (i.e., for about two months), when he had to give up work entirely. Meantime nothing seemed to relieve the pain and stiffness. His memoranda showed that by February 21 he was taking many doses of codeine and at night, almost regularly, doses of morphine, and besides these numerous doses of "bromidia" and still passing very sleepless nights. He had taken hot-air treatments, which seemed to benefit him at first, but were losing their effect. The foregoing gives an idea of the length and severity of the attack. When I first saw him, February 24, 1904, he was practically confined to bed. He had much difficulty in adjusting his head to the pillows in such manner as to obtain relief, and the getting-up and down process was very painful and tedious. There was an exaggeration of the same guarded carriage mentioned in Case 1. The head was not twisted and adducted to the painful side as in "inflammatory wry-neck," but was held rigidly in the middle line and flexed so that the chin was constantly depressed.

When asked to locate his trouble he indicated a point on the left side of the neck corresponding about to the transverse processes of the 4th and 5th C. From there the pain radiated through the occiput, to the top of the head and back of the ear, and downwards less severely over the cap of the shoulder to the insertion of the deltoid, and posteriorly to the spine of the scapula, i.e, conforming to the distribution of a neuralgia of the so-called cervico-occipital type. The most intense pain was usually in the occipital region and directly behind the ear. The paroxysms were distinctly worse at night. He often referred to a numb, unpleasant feeling in the ear (auricle), but there were no objective sensory changes here or elsewhere.

Manipulation of the neck was very painful, and it was impossible to gain much information in this way. It seemed, however, that there was articular as well as muscular resistance to movement.

He stated to me that about Christmas time of the previous winter he had experienced a slight attack of pain and stiffness in the neck, but it passed away in a few days. Several times during the following months he had felt little intimations of it, but he played tennis all summer in addition to his business duties and felt in excellent health until the time of the attack here described.

I could obtain no history of rheumatism, gout or syphilis. He had some scars, two large ones on his face, and a number of them widely distributed on the extremities and trunk. These he said were the remains of a "rupia" which had been treated three and five years ago, each time several months, by a physician in another city who had told him that the eruption was not syphilitic. A careful overhauling at this time and subsequently failed to divulge evidence of syphilis or other disease.

Inunctions of mercury were begun at once to the neck and to the back, and iodide of soda in increasing doses. At the end of two weeks there was much improvement. His appetite and strength were improved and the pains so much reduced that he was obtaining a night's rest on twelve grains of aspirin with one-half grain of phosphate of codeine taken once or twice.

March 30 (about one month after beginning treatment) notes were made as follows: Patient's head and neck movements rapidly becoming normal, and he cited various instances of the fact, e.g., in being able to again comfortably follow the ground in walking and to look to either side, and in stooping to pick up articles from the floor. He also volunteered the statement that he could extend the neck relatively better than he could flex it. In manipulating I was readily convinced of a sudden check to flexion between the 4th and 6th cervical vertebræ, and that it gave the impression of being articular instead of a muscular resistance. On the left side of the neck back of the tranverse processes of the 4th and 5th cervical vertebræ there was an area the size of a silver dollar still very sensitive and swollen to the extent that one could be quite certain of the tumefaction. Pressure here sent sharp pains through the neck, especially up to the occiput. On the right side of the neck opposite to this point there was also much deep tenderness, but not the sensitiveness that was found on the left. He still complained of a numb and at times stinging feeling in the auricle, but I could find no objective sensory changes there or elsewhere.

I saw the patient last in the latter part of May. There was still some sensitiveness on deep pressure and with extreme movements, but he was feeling perfectly well and following his usual active duties.

The bibliographic records of syphilis of the spine show that it was first recognized many years ago and that it is a rare affection. I find that the two cases here reported comport with what might be called a type of cases, a subclass of cervical spinal syphilis, characterized by a ragidly stiff neck with one or more points of great tenderness on deep pressure, severe neuralgic pains, often not very sharply localizable, no objective sensory changes, no paralysis, recovery on antisyphilitic treatment. Of course a diagnosis depending on this kind of data will be wrong at times, and yet at other times these symptoms alone may be grouped with such force as to leave little or no doubt. The question of differentiation, however, has been well covered by others and I will not dwell upon it. I simply mention it to show that cases of this kind have been discussed before and that they require close clinical observation.

A CASE OF TRAUMA OF THE FOOT OF THE SECOND FRONTAL CONVOLUTION, FOLLOWED BY ATAXIA, NYSTAGMUS AND EPILEPSY.[1]

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A. C.; aged twenty-four; mill-hand; single. Examined June 21, 1899.

Family History.—Mother, aged sixty-five; father, aged sixty-six. Both living and well. One brother, twenty-eight years of age, living and well. One sister, aged twenty-seven, rather delicate.

Personal History.—Had St. Vitus' dance as a child. Never had any serious illness except typhoid fever at twenty years of age. Made a good recovery. He was well at the time that the following injury was received.

On April 20, 1899, he went to Camden, N. J., to see a base ball game. While endeavoring to separate a number of men who were fighting, he was struck on the head with a base ball bat. He sought safety in flight, but while attempting to climb a fence, he received a second blow on the head. We was stunned, but did not become unconscious. He was taken to the office of a nearby physician, who sewed several extensive cuts upon the scalp. He was not removed to his home until the following day, when he came under the care of his family physician. He was unable to talk clearly, and finally became delirious. He was in bed for about five weeks during all of which time he was mentally much confused. Finally convalescence became established and he was able to leave his bed, but he continued to suffer markedly from headache and from difficulty in moving about.

Present Condition.—The patient presents two extensive scars in the frontal region. The first, which runs from before backward, begins just to the left of the median line, about one inch and a quarter from the edge of the scalp. It is an eighth of an inch in width and one inch and three-quarters in length. On the right side of the head, two inches to the right of the median line, a second scar begins, about one inch and a quarter in front of the border of the hair and running backwards. This portion constitutes the stem of a Y-shaped scar, running upwards horizontally for one inch and dividing into two branches. The branches of the Y are curved, the one running slightly upward and the other slightly downward. The second scar is by far the most extensive and apparently the most serious. The scalp is freely movable upon the subjacent bone. No depression can be discovered in the skull. Information received from the physician who attended the patient at the time of the accident is to the effect that no fracture was detected at the time that the scalp was dressed.

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[1] Read at the meeting of the American Neurological Association, September 15, 16 and 17, 1904.

The general examination yields the following results:

Station plus; sway exaggerated, possibly a little more marked in the lateral than the antero-posterior directions. The patient stands with great difficulty upon the right foot alone; cannot balance himself. Stands upon the left foot alone, but the difficulty of balancing is even more marked than when standing upon the right foot. The gait is slightly, but distinctively ataxic. The patient flexes the knees excessively, raises the legs too high and brings his feet down on the floor with a flop. The ataxia becomes more pronounced when the patient attempts to walk with his eyes closed. He presents a coarse intention tremor of the left hand. There is no tremor of the right hand. The dynamometer registers with the right hand 50; with the left hand 49. The movements of the left hand are decidedly awkward in executing the grip. There is no ataxia of the right arm. Coarse intention tremor or atactic movements of the left hand and arm are revealed when the attempt is made to touch the nose, chin and ear with the forefinger. There is no facial inequality. There is no paresis of any of the facial muscles. The tongue is protruded in the median line and is indented by the teeth.

An examination of the eyes fails to reveal any optic neuritis. There is, however, present a distinct nystagmus which is elicted when the eye-balls are rotated to either side. It is equally marked in both eyes. It is not a to and fro vibratory movement, but an irregular atactic movement. It is not elicited upon upward or downward movements. The pupils are equal and respond promptly to light. Reaction to accommodation and convergence is perhaps a trifle sluggish. There are no contractures of the visual fields.

The knee-jerks are slightly plus, but appear to be readily exhausted. They are equal. There is no ankle clonus. The bicipital reflex is present upon either side and equal. The elbow-jerks are also present and equal. There are no sensory losses; there is no loss of the muscular sense; no loss of the stereognostic sense. The patient complains of a dull feeling about the left angle of the mouth and adjacent portions of the upper and lower lips, but no actual sensory loss can be detected.

The patient sleeps well, but is nervous and easily upset. His hands are cold and damp. He suffers from palpitation of the heart. Has ringing in the ears and is dizzy. Hearing is somewhat impaired upon both sides. He hears an ordinary watch at a distance of two and a half inches from the left ear and five inches from the right ear. He adds, however, that he has always been hard of hearing. He also complains of a nervous sensation in the back of the neck and of pain and headache in the region of the scars.

Because of the intense headache and because of the ataxia and tremor of the left arm, an exploratory operation in the right frontal region was advised, but was negatived by the patient.

He now disappeared from observation and did not again present himself until September 12 of the same year. This time his condition was as follows: Station much impaired. Stands fairly well upon either foot alone. Gait still slightly ataxic, but much less so than upon former examinations. Swings his legs slightly forward and brings his feet down on the floor with a flop. Still has some intention tremor and ataxia of the left arm. The right hand registers with the dynamometer 72; the left hand 64. The pain and headache are much less. The knee-jerks are about normal. Eye symptoms unchanged.

Re-examined, December 20, 1899. Is not able to work because of lack of confidence in himself. States that he is liable to fall over toward his left side. On December 4th he had an attack of unconsciousness. He did not, however, have a convulsion. The right knee-jerk is slightly minus; the left knee-jerk about normal. His general physical condition is improved. Gait is much less ataxic, but he throws the left leg a little awkwardly still. The ataxia of the left hand is still present, but diminished. Says that his left arm feels almost as strong as before.

Re-examined May 4, 1900. Has worked for two months. On Easter Monday, April 16, had a second attack of unconsciousness. He was reading a newspaper when his mother noticed that he suddenly rose to his feet,, stretched out both of his arms, rolled his eyes and became unconscious. He also moved his head slightly to and fro. The patient states that he did not know that an attack was coming on nor was he afterwards aware that anything unusual had occurred, save from the statement of his mother. After this attack, however, he had severe frontal headache, and also felt very sleepy; he was very weak, especially from his knees down, and his mother put him to bed.

Re-examined as follows: Station slightly plus. Stands well upon the right foot but with difficulty upon the left. Some intention tremor of the left hand. Right hand (dynamometer) 73; left hand 58. Peculiarities of gait noted at previous examination still present. The left leg is still ataxic, and the patient complains of a sense of awkwardness in the left leg. Distinct ataxia of the left arm is also still present. There is in addition now a slight hypesthesia of the left arm. The knee-jerk of the left leg is now minus. The right knee-jerk is prompt, though not exaggerated. Eye condition as at first examination.

The patient now finally consented to the oft-repeated advice to submit himself to surgical interference, and on May 24, he was operated upon by Dr. W. W. Keen.

Dr. Keen turned down a large osteoplastic flap in the right fronto-parietal region, the anterior border beginning just at the border of the hair, the posterior somewhat back of the fissure of Rolando. The dura toward the anterior portion of the opening was distinctly yellowish in color and the brain was softer to the touch than the posterior portion. The dura was now turned down and found over an area of about 4 or 5 cm. adherent to the pia mater so that it required considerable force to separate it. As a result of this separation, several vessels had to be ligated to check the hemorrhage. Pressure at this portion of the brain for the purpose of arresting hemorrhage produced very distinct pitting, such as one sees over the tibia in edema of the leg. In order to determine whether there was any cyst or other abnormality, Dr. Keen punctured the brain at a depth of 3 cm., but found nothing. An incision into it showed that the cortex was considerably darker and redder in color than normal. The dura was then closed with silk and the flap with silk-worm-gut. The region of the cortex especially involved was evidently the posterior portion or foot of the second frontal convolution. The patient made a perfectly smooth recovery; only one day was the temperature above 100°. He went home June 1, a week after the operation, the wound being entirely healed.

He was re-examined June 25, 1900. Pain and headache had entirely disappeared, but the general physical signs were the same as previous to the operation. There was still present a slight ataxia of the left arm and left leg. Both knee-jerks were minus. Nystagmus was still present upon deviation to either side.

October 22, 1900, the patient again presented himself. He had had an epileptic seizure three days previously, the first since the operation. The attack was attended by unconsciousness and as far as could be learned, the convulsive movements involved both arms and both legs. Strange tickling sensations persisted at the site of the old injury for some time after the attack.

He presented himself subsequently at intervals of two or three months, until January 11, 1902. The epileptic seizures had not recurred. All trace of ataxia in the left leg had disappeared, his gait and station were entirely normal. Ataxia also had almost entirely disappeared from the left arm. Lateral nystagmus, however, was still present in both eyes. No plantar reflex could be elicited upon either side.

He was last seen by me on July 9, 1903, when the following note was made. Ataxia is still absent. He stands very well upon either leg alone. Both knee-jerks normal at present. Has a disappearing intention tremor in the left hand. Slight awkwardness of movement is still noticed in the left hand in executing movements of precision. Still has nystagmus on lateral deviation toward either side, more marked in conjugate deviation to the left. There is no astereognosis. No feeling of numbness in the hands. Patient complains that he is unable to do hard work. Has a sensitive spot in the right temple. Has not had an epileptic attack for over a year. Is learning cabinet and pattern making. Is very seldom troubled with headache, but often has ringing in the ears.

The above case is especially interesting because of the presence of ataxia and nystagmus. It is well known that tumors and other lesions of the frontal lobe may give rise to ataxia, an ataxia which is sometimes bilateral, as in the present case. Attention was years ago directed to this fact by Bruns, Oppenheim, Wernicke and others. As regards the nystagmus or atactic movements of the eye-balls, the fact that the second frontal convolution is a center which stands in direct relation to the eye movements is most suggestive. Ferrier, Beevor and Horsley, Mott and Sherrington have demonstrated the associated eye movements of this region experimentally. Conjugate deviation and turning of the head have also been noted in man in lesions of this region. Nystagmus or ocular incoordination had not, however, been observed. Just as this paper was being prepared, a report of a similar case by Klien appeared in the *Zeitschrift für Nervenheilkunde,* Vol. 26, 1904, page 327. Klien places upon record a case of incoordination of the eye movements after trauma of the head, producing a superficial lesion of the foot of the second frontal convolution. The symptom observed was especially that of horizontal nystactic movements. General ocular incoordination, however, appears to have been present for while almost continuous horizontal nystactic movements occurred upon attempting to use the eyes, the attempt to fix the eye on a definite point produced irregular movements in the most diverse directions. Among others pronounced slow rotary movements were observed. Further in Klien's case, the right eye presented these disturbances of motion in greater degree than the left;—the trauma was on the left side of the brain. In my case the movements were present in equal degree upon the two sides. In Klien's case it was especially observed that both eyes acted extremely independently of each other. Nothing of the kind was observed in my case. In Klien's case on the other hand, there was no ataxia of the arms or legs and no epilepsy. There was, however, decided loss of sensation upon the entire right half of the body both to touch and pain. The right extremities were somewhat paretic, the right arm more than the right leg. While the two cases resemble each other, they were not therefore exactly parallel.

It is interesting to note that while the ataxia of the extremities in my case finally disappeared, the nystagmus steadily persisted. Had these symptoms been present in a lesion not caused and not readily localizable by the trauma, the ataxia and the nystagmus would have at once directed the attention of the observer to the cerebellum. It must perhaps be admitted that trauma causing a lesion of the frontal lobe might also cause a lesion of the cerebellum. This possibility must, however, be regarded as a remote one. It is significant further that while the ataxia was bilateral, it was much more marked in the left leg and persisted for a much longer period than in the right, the trauma having taken place in the right frontal region.

The case also teaches a valuable practical lesson as regards the convulsive seizures. These were general in character and not focal. The patient, at the beginning of an attack would rise to his feet and extend both arms; that is, there would be a simultaneous disturbance of the innervation of both halves of the body, although the lesion giving rise to the disturbance was a limited one. That this lesion was the cause of the seizures is proven by their disappearance after the surgical operation. The logical inference is that in lesions of the foot of the second frontal convolution, which give rise to epilepsy, the epilepsy is not necessarily focal. Probably this inference applies to other portions of the frontal area as well.

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

November 1, 1904.

The President *pro tem.*, Dr. Joseph Fraenkel, in the Chair.

Two Cases of Bilateral Symmetrical Atrophy of the Thenar Eminence.—These were presented by Dr. I. Abrahamson. The first patient was a woman, sixty-two years old, whose family and personal history were negative. Her occupation was that of a housewife, which necessitated putting her hands in water a great deal, and she also assisted her daughter at needlework. Eight years ago she first began to experience numbness and paresthesia in both hands, with beginning atrophy of the thenar muscles, chiefly the flexor brevis pollicis. There were no trophic skin changes and no nerve tenderness. The atrophy was symmetrical in both hands.

The second patient was a woman, fifty-two years old, a native of Hungary. Her occupation was that of a housewife. The symptoms were similar to those in the first case, but the paresthesia was most annoying at night. There was marked symmetrical atrophy of both thenar eminences. The speaker said he regarded both of these cases as examples of acro-paresthesia with subsequent atrophy.

Dr. Charles L. Dana said the unusual feature of these cases was the symmetrical character of the atrophy affecting both hands. Not long ago, the speaker said, he saw a case of atrophy of the thenar eminence of one hand, with paresthesia and evidence of neuritic disturbance. In this instance the condition had been produced by the Vardon grip in playing golf. In another case the patient was an elevator boy, and the neurosis had been produced by pulling on the wire ropes of the elevator. While these occupation neuroses were not particularly rare, bilateral and symmetrical cases of this kind were extremely so.

A Discussion on the Classification of the Melancholias.—Dr. Charles L. Dana opened this discussion. He stated that the term melancholia indicated to the psychiatrist a combination of symptoms varying in groupings, duration and intensity, but beneath all a dominant condition of painful emotional depression. It might occur as a symptom in various organic and degenerative cerebral diseases, but it was recognized in particular as a manifestation of two functional states: first, that occurring at about the period of involution, or change of life, and later: second, that occurring in early and early middle-life. No one, the speaker thought, disputed the distinctive character of melancholia of the first type as it occurred in middle and late life. The "melancholia of involution" was a chronic and incurable disorder. It was a psychosis accompanied with anxious, worrying apprehension, and was often a natural evolution of the constitutional worrier. It was characterized by hypochondriacal and obsessive ideas, dysthesia and somatic delusions; by hallucinations, self-accusations, and at times by suicidal ideas and impulses. There was complete self-absorption, narrowing of consciousness, enfeebled attention and enfeebled power of concentrated or constructive thought. There was no clouding of consciousness nor retardation of thought nor dementia. It was an agitated, worrying hypochondriacal psychosis.

The melancholias which were seen, especially during early life, and were at least characteristic of that period, had not such a single, definite picture. That form which had been described as acute melancholia, however, was in its typical form a sharply defined condition, characterized by profound emotional depression, retardation and difficulty of thought, clouding of consciousness and other phenomena familiar enough to all. This form of melancholia, we were told by Kraepelin and his followers, was not really melancholia, but only the melancholic phase of manic-depressive insanity, and it was proposed to substitute this term for all or most of the functional melancholias not belonging to the melancholia of involution. Hence we would have but one real melancholia, the chronic form of later life.

Dr. Dana said he had been trying to fit this scheme to his experience, and for that purpose he had looked over the histories of over 400 cases occurring in his private practice. Of these, he had closely analyzed 130, which he had seen more recently and in many instances followed for a long time. From this analysis he had reached the conclusion that chronic melancholia, under the classification of Kraepelin, was indeed a special disease, but not in all cases one of the involutional period. In its milder types especially it seemed to him that we saw pictures of it that would be called anxiety psychosis, or depressive neurasthenia, and it was not unreasonable to suppose that in psychopathic individuals, or those with a special hereditary tendency, the nervous system should be subject to early chronic melancholia. He thought it possible that this same worrying psychosis might appear at the period of adolescence, when the disease was less intense, the somatic and sensory disturbances less severe, but the mental state essentially the same. The sharply defined delusional melancholia of early life, with great activity of thought, clearness of memory, intense self-accusation and powerful suicidal impulses belonged more to this type than any other. The association and alternation of mania with early, simple recurrent melancholia was an established fact, and the term manic-depressive insanity fitted this group of cases admirably, but in the speaker's list of 400 cases of melancholia, mostly not asylum types, there was a history of mania in only 8 per cent. It seemed rather forcing the issue, therefore, to use the term manic-depressive insanity for these patients, who never had any mania. He was inclined to regard the melancholias of early life as simple melancholias of the involution type—or preinvolutional, if that seemed a better form. He would at least consciously associate their disorder to that form seen more typically and severely later in life. He would say that there were a great number of simple, functional, often recurrent melancholias that belonged to the manic-depressive type by reason of the neurasthenic and apathetic condition, the absolute loss of interest in life, and psycho-motor sluggishness and enfeeblement, but he saw no reason to use the term manic-depressive insanity here. He would use the term manic-depressive insanity only when there was evidence of it.

Dr. M. Allen Starr said that Dr. Dana's remarks regarding melancholia would appeal to every neurologist and practitioner who did not constantly come in contact with the severer types of insanity. Those who were doing asylum work, however, might meet with considerable difficulty in grouping certain of the cases. Perhaps 5 per cent. of the cases in the average asylum could not be positively assigned to any definite group. The speaker said he was glad to hear Dr. Dana make a distinct protest against the term manic-depressive insanity. He had never been in sympathy with that term, for the simple reason that it did not coincide with our clinical experience in this country. He had carefully reviewed the records of about 280 cases that he had classified as melancholia, and he saw no reason to protest in any way against the classification that was proposed by Krafft-Ebing years ago, namely, simple melancholia, melancholia with delusions, and melancholia with delusions and agitation. These three types embraced distinct varieties of the disease. The speaker said he also agreed with Dr. Dana that the type that rose to the stage of mania was extremely rare, in his own experience, probably not exceeding 5 per cent. of the total number of cases observed. He thought it would be very unfortunate, therefore, to label these patients with a term like manic-depressive insanity, in spite of the fact that in 95 per cent. of the cases there was no mania whatever. Dr. Starr said that a careful study of his cases of mild melancholia had impressed him with the idea that the condition was a purely toxic neurosis. The symptoms were most severe in the early morning, and passed off largely during the day. Sixty per cent. of his cases never passed beyond the simple melancholic type, which was not the type met with in the insane asylums. The cases he had in mind were entirely conscious of their surroundings and of their own condition; they were in extreme dread of insanity, and they were not suitable subjects for an insane asylum, and to incarcerate them in such an institution would be an outrage. About 35 per cent. of his cases corresponded to the delusional type, and required watching to prevent suicide.

Dr. Starr said he would like to impress upon the Society the existence of a true toxic melancholia. In fully 60 per cent. of his mild cases he believed that he had to deal with a purely toxic neurosis, and if we could eliminate the poison, we could cure the patient. He had noted that any undercurrent disease, such as grippe, pneumonia, or malaria, that produced a decided change in the chemistry and nutrition of the body, arrested the symptoms in these mild cases of melancholia, and he had produced the same effect by the use of thyroid extract. Under the administration of this agent the symptoms of melancholia would be immediately relieved and remain absent until the temperature again dropped to normal.

Dr. Adolph Meyer said that his experience with the melancholias had naturally been of a different sort from that of Drs. Dana and Starr, having been largely confined to patients who were already committed to the asylum. His observations had led him to conclusions which did not essentially differ from those of Dr. Dana, although for practical reasons he would rather favor a different classification. On the whole, he was desirous of eliminating the term melancholia, which implied a knowledge of something that we did not possess, and which had been employed in different specific ways by different writers. If, instead of melancholia, we applied the term depression to the whole class, it would designate in an unassuming way exactly what was meant by the common use of the term melancholia; and nobody would doubt that for medical purposes the term would have to be amplified so as to denote the kind of depression. In the large group of depressions we would naturally distinguish our cases according to etiology, the symptom-complex, the course of the disease and the results. A distinction into acute and chronic forms is not consistent with experience. Dr. Hoch finds that all attacks tend to become longer in advanced years. The distinction had best be made according to the intrinsic nature of the depression. From that point of view we might distinguish the pronounced types from the simple insufficiently differentiated depressions. Besides the manic-depressive depressions, the anxiety psychosis, the depressive deliria and depressive hallucinations, the depressive episodes of dementia præcox, the symtomatic depressions, non-differentiated depressions will occur; especially the differentiation of the dementia præcox type frequently causes difficulty, where stupor and catalepsy supervene. The speaker said he was somewhat surprised that no reference had been made to the difficulties in determining the depressions of dementia præcox. The speaker took exception to Dr. Starr's statement that mania was but a rare event and only to be expected in the most agitated cases. He could not recall a single instance where a case of the pure agitated type developed mania, while on the other hand, the type that did not show the agitation, but a great deal of inhibition and subjective inadequacy, was the form that might develop mania.

Dr. Meyer said that he would rather hesitate to accept Dr. Starr's idea that the intermission in the symptoms of melancholia produced by febrile disturbances proved the toxic origin of those symptoms, because an intercurrent disease was apt to produce a change in the symptoms of almost any mental disorder. The shortness of the time for discussion did not permit the review of about 300 cases of depressions to be reported on another occasion.

Dr. Ralph L. Parsons said that some years ago he made out a classification of the insanities that was provisional only, because the facts upon which to construct a scientific classification did not exist then nor do they exist at the present time. Different types of insanity were often very closely identified, and during the same attack the patient might exhibit symptoms of mania, melancholia and dementia. Dr. Parsons then exhibited a table that he had devised, in which the melancholias were divided into many different types descriptive of their cause, such as adolescent, puerperal, etc.

Dr. Joseph Collins said that while some writers on mental diseases claimed that melancholia was a very uncommon disease, and much less frequently met with to-day than mania, others maintained that there was no such disease as melancholia, and that that which we called melancholia was a symptom of a diagnosticable condition, which, being the real disease process, should be entitled to the name. Dr. Collins said he was in sympathy with the latter writers, in so far as their position compelled them constantly to seek for the dependency of melancholia, but he was unwilling to accept their statement that there was no such disease as simple melancholia, i.e., melancholia which was not a constituent of other disease, physical or mental. The general practitioner was likewise loath to accept such statements, because they seemed to him to be at variance with his experience. The difficulty in discussing the subject was that the term melancholia was used by writers with great latitude and absence of specificity.

Melancholia, or mental depression, coupled with moral insufficiency, sadness and anguish, was of common occurrence. It was encountered in certain toxic states, such as alcoholism, and as the result of insufficient oxidation and deficient elimination. It was found associated with neurasthenia, hypochondria, paresis, general and cerebral arteriosclerosis, dementia præcox and mania, and also occuring without any of these associations. Every neurologist saw a great many cases which he diagnosticated as melancholia without being able to satisfy himself of the dependency of the melancholia, just as the general practitioner or consultant in internal medicine must often still make the diagnosis of indigestion without being able to satisfy himself that it was dependent upon any one or more of a number of disordered conditions that were sufficient to cause it. When the neurologist had the opportunity of observing these cases for a protracted time, he was enabled to classify them more specifically, and from an etiological standpoint more correctly. There was a tendency at the present time to put all cases of melancholia that could not be referred to organic disease and manic-depressive insanity under the head of "involution melancholia," i.e., melancholia associated with retrograde changes in the tissues, and occurring at a time of life when such changes of the tissues set in physiologically, and therefore are directly or indirectly dependent upon them. It was the writer's experience that considerably less than one-half of all the cases diagnosticated as melancholia, using the term in the specific sense mentioned above, could be placed in this category.

Dr. Collins then cited in detail a number of cases of melancholia which illustrated the great variation in the onset, delineation and course of the disease, and the difficulties that beset the attempt to place them in one or two groups. The neurologist often had much difficulty in deciding whether such a case should be called neurasthenia, hypochrondria or melancholy depression. Some of these patients eventually became insane (using the term in its legal sense), but he believed that the vast majority of them did not, nor was their mental capacity so interfered with that they had to abandon their business. Whether or not there were such a disease as acute melancholia was of the greater importance, because it carried with it the matter of prognosis. The outlook in manic-depressive insanity and involution melancholia had been fairly well determined, and the diagnosis of either one carried with it a serious prognosis.

Dr. Collins said he agreed entirely with Dr. Dana, excepting in his statement that there were two types of melancholia with three names for them. There were three types of melancholia, namely, the acute, the manic-depressive and involution melancholia. So far as the manic-depressive type was concerned, the speaker said he knew of no disease with more clearly drawn clinical features, none which could be more readily recognized, and none in which the etiology, course and eventuation were more certain. He saw no more reason for eliminating manic-depressive insanity than he did for eliminating appendicitis, streptococcus meningitis or other well recognized conditions.

As to the relation of toxic conditions to melancholia, Dr. Collins said he had never seen a case of melancholia that was relieved in any way by medication directed towards the supposed toxic agent, and the very fact that the symptoms became intermittent or were held in abeyance by the administration of other poisons, or by an intercurrent attack of grippe or pneumonia, would seem to militate against the contention that they were of toxic origin. Otherwise it would be a strong argument in favor of the doctrine of *similia similibus curantur.*

Dr. B. Sachs said that in a discussion of the classification of the melancholias there was danger of going to extremes in both directions, and making the classification either too wide or too narrow. Under the influence of Krafft-Ebing and the older French and German writers the term melancholia was much abused. It should be restricted, he thought, to those cases in which there was a slowing up of all mental processes, associated, perhaps, with some self-accusation. This formed a very definite clinical picture, and he saw no reason why it should be eliminated. On the other hand, there were various forms of depression which could be differentiated from melancholia. In private practice he frequently made the diagnosis of simple depression; this he regarded as a transitory condition, and it had never occurred to him to look upon it as true melancholia, which was a true and very often a serious psychosis. To his mind, pure melancholia had always represented a typical and grave disorder. The question arose whether to discard the older classification entirely in favor of the more recent one of Kraepelin, with its comprehensive term of manic-depressive insanity, which included all types of melancholia, circular insanity, and what not. In picturing manic-depressive insanity, Kraepelin had shown great genius and insight, and Dr. Sachs said he indorsed that writer's wisdom in putting clearly before the medical public the existence of a large group of cases that were characterized by typical symptoms. There could be no doubt about the existence of such a group, but he could not agree with the dictum of Kraepelin that all cases of melancholia are merely one stage of manic-depressive insanity.

Dr. A. R. Diefendorf, of Middletown, Conn., said he was surprised by the difference of opinion regarding the melancholias as expressed by the neurologists and those who saw these cases in asylums, as it was certain that all the cases that eventually came to the hospitals for the insane had passed through the hands of the practitioner and specialist. As regards simple melancholia, there were several groups of those cases. There was one group in which the characteristic symptoms were mental sluggishness, a feeling of insufficiency or inadequacy, a lack of energy, an increased sense of fatigue, a slowing of the train of thought, difficulty of comprehension, perhaps a few indefinite hallucinations and possibly a few delusions. The emotional attitude of these patients was one of simple despondency. They took an abnormal view of life. There was another type of simple melancholia that occurred early in the course of senility.

Dr. Edward D. Fisher said he would certainly agree with the view that we had to deal with a simple melancholia which was distinct from that described by Kraepelin. Kraepelin's cases were those of later life, and differed from the melancholia which he found in manic-depressive conditions. There was in his so-called melancholia not so much absolute retardation of thought as in the class of cases of the manic-depressive type. Dr. Fisher said he had seen a number of cases occurring in young persons, perhaps about the age of twenty-five, where they simply presented a psychosis, which was described as melancholia, and where the patients made a complete recovery within three to six months. He could recall one case in which at least fifteen years had elapsed since such an attack, and there had been no signs of a recurrence. The speaker said he was certainly in favor of retaining the term simple melancholia, and he also believed decidedly in the manic-depressive type.

Dr. L. Pierce Clark said he was not in favor of the term involution melancholia. He thought the psychosis of the climacteric or the melancholia of involution should more properly be called an anxiety psychosis, because that seemed to be the great predominating factor. In the differential diagnosis of manic-depressive insanity and involution melancholia the one essential feature was the retardation.

Dr. A. J. Rosanoff said it was well known that depression was the most common symptom encountered among the insane. He thought that all forms of mental disease, without a single exception, might present this symptom. This fact perhaps accounted for the statement of the older writers with regard to the frequent occurrence of "melancholia." At the institution with which he was connected, and probably also at all other asylums, a considerable proportion of the depressed cases admitted had to be set aside as unclassified. The majority of these cases ultimately developed one or the other of the typical syndromes, but a small group still remained which could not be classified among the well-known psychoses. This group contained about five per cent. of the total number of depressed cases admitted to the asylum. This was merely a rough estimate, but the speaker said he felt assured that it could not be far from the actual figures. It had been proposed to designate these cases as "simple melancholia." It seemed to him that any generic term would be entirely inapplicable to an extremely heterogenous group of psychoses, such as these.

With regard to manic-depresssive insanity, Dr. Rosanoff said it must be remembered that many cases presented during their whole life nothing but a number of attacks of simple depression. The diagnosis was made not at all from the occurrence of manic and depressive episodes in the same individual, but chiefly from the exceedingly well-defined symptomatology. He thought that Kraepelin had selected this term for the disease for the purpose of indicating simply that its manifestations might be either depression with retardation and dearth of ideas, or elation with restlessness and flight of ideas, or a mixture of these phenomena, but not that each individual case must present all the possible manic-depressive episodes. Some of the depressed cases of this disease were so mild that the patients were not committed, and were even able to continue their occupation; but even those cases presented the characteristic features of the disease, and one experienced in making the diagnosis of manic-depressive insanity would not have much difficulty in recognizing them.

The occurrence of melancholia with anxiety and agitation at a comparatively early age had been referred to. The resemblance between these cases and those of involution melancholia of Kraepelin was only a superficial one. The speaker thought that probably almost all of them belonged to the dementia præcox group. On analysis their depression was found to be extremely shallow, it being at times but a simulated depression. Moreover, they presented mannerisms, stereotypy, neologisms, negativisms, emotional deterioration and delusions of an exceedingly absurd or even impossible nature, contrasting with perfect orientation, intact memory for recent and remote occurrences, and a surprising alertness with regard to their surroundings.

Dr. Dana, in closing, said that on the whole, the position he had assumed in his paper had been largely sustained by those who had taken part in the discussion. He still insisted that we have two forms of melancholia, with a third that belongs more or less to one or the other. The two forms are the involution type and the manic-depressive. Outside of these there are many cases of simple depression, which he had learned from experience would never be anything else but simple depression, but which, if closely analyzed, would be found to belong to either the manic-depressive or the involution type. In his paper, the speaker said, he had confined himself to the functional types of melancholia, disregarding the group of simple symptomatic melancholias occurring in the course of organic brain disease. The classification devised by Dr. Parsons was very practical and interesting, especially for those working in institutions, but, of course, it was not intended as a text-book guide of melancholia.

A Case of Bilateral Facial Hemiatrophy.—Presented by Dr. Edward D. Fisher. The patient was a young girl who, about two years ago, had an attack of persistent vomiting. Following this, progressive wasting of both sides of the face was noticed. There seemed to be no atrophy of the bone, but absolute loss of subcutaneous tissue. There were no sensory disturbances; no electrical changes.

Dr. B. Sachs said the case was unique, so far as his experience and reading went. It differed from the ordinary facial hemiatrophy in the wide distribution of the lesion, and the absolute disappearance of the subcutaneous fat without involvement of any of the other tissues. It was certainly not a scleroderma. The girl's body was apparently well nourished.

The Nature of the Nerve Lesion in Brachial Birth Palsy (Erb's Type).—Dr. Thomas P. Prout read a paper on this subject. He stated that in order to properly appreciate the pathology underlying brachial birth palsy (Erb's type), the etiology of the over-stretching process in its production must be borne in mind, in sharp contradistinction to the oft-repeated statements in the text-books that it is produced by direct compression of the nerve trunks between the clavicle and the transverse processes of the vertebræ, or the clavicle and first rib. It is possible that the Erb syndrome may be produced in this manner, but when it occurs the behavior of the case is very different from that observed in the cases we are here considering. Pressure lesions in nerve bundles, if uncomplicated, recover rapidly, whereas in these cases we have some condition of a permanent character in the nerve bundle which prevents complete recovery. The cases in which the only etiological factor is pressure, recover within a year or eighteen months. The cases we are here considering reach a certain stage of recovery, and then present a permanent palsy of greater or less extent. What pathological lesion renders these cases permanent?

The behavior of the perineural sheath (a dense, connective tissue structure surrounding and supporting the nerve strands) during the process of repair is of great importance. Since this structure supports the nerve strands its rupture occurs as a primary factor in the cases we are here considering. In fact, in any nerve lesion produced by an over-stretching process, this supporting sheath must first give way. In these cases it is torn asunder and the arterioles belonging to it and supported by it are ruptured. A hemorrhage into the substance of the nerve and its sheath results. These facts have not heretofore been recognized in the literature of this subject. They are of the greatest importance, since they determine the ultimate extent and final character of the lesion. Were it not for the obstructive features of the repair process in the nerve sheath, we might expect a more or less complete recovery in the vast majority of instances without operative excision and suture.

Photomicrographs were shown of a case in point. (In all four cases have been examined to date.) In this instance only the fifth and sixth cervical roots were involved at the usual seat of the lesion, their junction to form the plexus. The perineural sheath presented many old, dense pigment deposits, the site of old hemorrhages. In some portions the perineural sheath was buckled inward upon the nerve fibers, strangulating them and preventing their regeneration. Evidences of strangulation were not only present at these points but also in the nerve fibers underlying the pigment deposits. There was obliteration of the myelin sheath immediately beneath these areas, and fragmentation of the myelin sheath above and below. In the more severe cases the strands of the plexus involved came to an abrupt termination in a mass representing an old organized hemorrhage. In these instances there was a severing of the nerve fibers, which were often thrown into folds for some distance from the site of the primary lesion.

The importance of these lesions cannot be overestimated. Repair of the nerve sheath takes place before regeneration of the nerve fiber, and if this has buckled inward upon the nerve bundles following the relief of tension, the nerve fibers are inevitably going to be strangulated and their regeneration prevented. The same applies to the organization of an old hemorrhage impinging upon nerve strands. The nerve fiber may or may not be ruptured. However this may be, the pressure of the old hemorrhage and the cicatrix in the perineural sheath are sufficient not only to prevent regeneration in the severed nerve fibers, but to determine a neuritis in those not severed, and prevent their regeneration.

The sequence of events in the production of the obstetrical birth palsies may be summarized as follows: The lesions are (1) immediate and (2) remote. The immediate lesion consists in a tearing of the perineural sheath surrounding and supporting the nerve trunk, and the incidental rupture of the blood-vessels belonging to it. Hemorrhage occurs into and beneath the perineural sheath. There is, furthermore, a severance of the nerve strands, more or less complete, depending on the severity of the case.

The remote lesion is brought about, and its extent determined by (a) the healing of the perineural sheath; (b) the organization of the blood-clot; (c) the ultimate contraction of the cicatrix upon the nerve strands, which not only prevent their regeneration but determine a pressure neuritis in those not severed upon which it may chance to impinge. This factor is of very great clinical importance. An infant in whom this accident occurs, who remains more or less continuously peevish and fretful for some time afterwards, and in whom the handling of the extremity greatly increases its pain and irritability, is suffering from a traumatic neuritis, due to pressure upon certain nerve strands of the organizing blood-clot and the contraction of the cicatrix in the perineural sheath. Cases that do not present this symptom have been produced by simple trauma to the nerve, without rupture of the perineural sheath, and will recover spontaneously, while in those cases presenting this sign of a neuritis a considerable palsy will follow, depending on the severity of the lesion. These facts have an important bearing on treatment.

Dr. Sachs said that a certain number of brachial birth palsies of the Erb type were due to pressure and nothing else. A large proportion of the cases were absolutely painless; there was simply numbness, but no pain, and no symptoms such as we would associate with a laceration of the nerve. The speaker thought that Dr, Prout took an extreme position in claiming that some of these cases, at least, were not due to pressure, and that laceration of the nerve was necessarily the cause of this type of birth palsy. There was no doubt that in some of the violent cases the nerve strands were torn, but in the large proportion of cases he thought the symptoms were the direct result of pressure and hemorrhage.

Dr. L. Pierce Clark thought from the data which he and Dr. Prout had at their command that in the vast majority of instances the palsy was the result of stretching and laceration and not of pressure. This was clearly demonstrated in the operative cases. In these the recovery of the patients was uneventful, and the progress very satisfactory. The pathological work was particularly interesting, in so far as nothing like it had ever been done in connection with this type of palsy.

Dr. William M. Leszynsky said he understood from the paper that in those cases that did not undergo spontaneous recovery, surgical interference was necessary. That being the case, he assumed that Dr. Prout concluded that in all cases that recovered without operation spontaneous regeneration of the nerve had taken place, and that those cases, therefore, had no lesion of the kind he described. The question therefore arose, when should we operate on these cases, and how could we know whether spontaneous regeneration would take place or not?

Dr. Prout, in closing, said that if spontaneous regeneration occurred it was not necessary to operate. This could be fairly definitely known within a year or eighteen months after the birth of the child. The cases that did not recover spontaneously presented exactly the lesions he had demonstrated, which represented a condition in the perineural sheath of the nerve which prevented spontaneous recovery. It was well known that many cases recovered spontaneously, but those that did not were due to stretching and not to pressure. Those due to the latter cause would in all probability recover spontaneously. If they did not, however, we were not dealing with a pure pressure palsy.

PHILADELPHIA NEUROLOGICAL SOCIETY.

October 25, 1904.

The President, Dr. C. S. Potts, in the Chair.

A Case of Hysteria.—This case was presented by Dr. Luther C. Peter. Hannah H., thirty-two years of age, a clerk, enjoyed good health until three years ago, when she gradually lost power throughout the body, staggered in walking, and in the course of a few weeks became bedridden and paralyzed. After months of treatment she improved, and was able to walk with difficulty. She now complains of unsteadiness of gait, great muscular weakness, a girdle sensation, severe at times, about the waist, and a sensation of walking on air. Sphincters are continent.

She is a small woman, of poor musculature; her gait is a mixture of ataxia, spasticity and muscular weakness. The ataxia in arms can be overcome by practice, and to some extent the same is true of the ataxia of the lower extremities. All deep reflexes are increased but variable, while clonus is present at times, and at other times there is only an attempt at clonus. Babinski's reflex is present on both sides; station is bad with eyes open, worse when closed.

There is hypesthesia in upper part of chest, but touch sense is preserved in the rest of the body. Thermic and pain senses are lost over the entire body beneath the middle of the thorax, except over right trocanter and in the region of Scarpa's triangle on the right side. Above the mid-thorax there is hypesthesia in pain and temperature senses. She cannot taste, and the faucial reflex is absent. The eye-grounds are normal. Both visual fields are concentrically contracted, and there is inversion of the red and green color fields. Cornea and sclera on the right side are anesthetic in the lower half, and in the upper half on the left side.

Although the Babinski reflex is present, it is the only constant symptom which points to organic disease. An early diagnosis of disseminated sclerosis plus hysteria was made, the sensory and eye phenomena, the peculiar form of ataxia which seems to be in a measure under the patient's control, the variability in the symptoms and the improvement, which, though slight, is appreciable, have led to a diagnosis of hysteria.

Dr. Mills stated that the patient had been under his care at one time at the University Hospital. He had examined her several times very carefully, and became satisfied, after she had been under his observation for some time, that the case was one of organic disease, notwithstanding the fact that she presented a few of the classical symptoms that are supposed to clearly indicate the presence of hysteria. He did not believe the contractions of the visual fields are undoubted evidence of hysteria, as they are known to occur in other diseases, and sometimes in health. He stated that the patient did not seem to be any better than when he had first seen her, the ataxia of station and gait being no less marked. She still has a marked, persistent ankle clonus on the right side, and clonus on the left side, which is not so persistent. She has, also, the Babinski reflex on both sides. He stated that if the case were not one of pure hysteria (and he believed it was not) it more closely resembled a case of disseminated sclerosis. He believed it to be one of organic disease, with associated hysterical symptoms.

Dr. Gordon thought that on account of the patient's gait being undoubtedly one of ataxia paraplegia, the marked spasticity, the persistent ankle clonus, and the presence of the Babinski reflex, functional disease could be eliminated. He called special attention to the presence of the Babinski reflex on both sides. According to Babinski's researches and the consensus of opinion of the Paris Neurological Society, when this reflex is persistently present, the case must be organic and cannot be functional. He also thought the presence of ankle clonus with the other symptoms, pointed almost certainly to organic disease. While the patient does present symptoms of hysteria, he believed them to be associated symptoms.

Dr. Spiller stated that he had seen and lectured on the patient at the Woman's Hospital about three years ago; that she had been under his care a long time at the University Hospital, and he was not surprised that a diagnosis of hysteria had been made. He believed she had improved somewhat, but never to a marked degree, and he had long since given up the diagnosis of pure hysteria in the case, and believed it to be a case of disseminated sclerosis, with hysterical manifestations.

Dr. Dercum believed the case to be one of organic nervous disease, and although the patient had hysterical symptoms he thought it very unlikely that the case was one of hysteria alone.

Dr. Peter, in closing the discussion, stated that he had brought the patient before the Society because he thought the case was of sufficient interest to provoke discussion. When she first came to the clinic she was thought to have disseminated sclerosis, plus hysteria, but after examination from week to week he felt that the hysterical element predominated. Even though the Babinski reflex was present, it seemed to him that the case was one of hysteria only.

A Case of Trauma of the Cervical Spine Exhibiting Syringomyelic Symptoms.—This case was reported by Dr. J. H. W. Rhein.

Dr. Alfred Gordon said that Dr. Rhein's case was identical in its clinical manifestations with the case exhibited by him (Dr. Gordon) before the County Medical Society in June, 1904. His was one of gunshot wound in the neck; bullet penetrating at the level of fifth cervical vertebra. The patient presented a motor paralysis on one side and a sensory paralysis on the other. Both conditions covered an entire half of the body. Dr. Gordon made a diagnosis of Brown-Séquard's paralysis, as it reminded him in the main features, as well as in detail, of the experimental results obtained in animals by that great physiologist. He, therefore, believed Dr. Rhein's case to be one of Brown-Séquard's paralysis.

A Case of Myokymia with Remarks Upon the Different Forms of Tonic and Clonic Myospasm.—This case was presented by Dr. C. K. Mills. The muscular twitchings are not always present. The patient is twenty years of age and very tall, over six feet, and has grown very much in the last two years. He is a student. One year ago he suffered with pain in the back of the head and neck, and had difficulty in concentrating his mind on his work. He also became easily exhausted physically. About this time he noticed at intervals muscular twitchings in different parts of the body, in the calves of the legs, thighs and the muscles of the shoulders, and between the shoulder and arm. These twitchings are so marked that he can not only feel them but can also see them. Formerly they were cramp like, but this is not the case any longer. He has no other symptoms except attacks of dizziness or a feeling of faintness on exertion. He has always been thin. The question of diagnosis was between fibrillary tremor or some peculiar form of cramp-like movement, or the so-called myokymia or myospasm of unknown pathology, usually supposed to be functional and connected with a neurasthenic state.

Dr. Dercum thought the whole subject of myospasm a very obscure one. A large number of myospasms do not seem to be related at all to fatigue symptoms, and again there is another group in which fatigue symptoms are a pronounced factor. He cited the case of a young school teacher in whom the condition was present in both arms. There was a prolonged period of fatigue before the muscle spasms became pronounced. At the present time her condition improves with rest and becomes worse with fatigue. Fatigue myospasms of the extremities are probably related to the muscular twitchings observed in neurasthenic states in the muscles of the calves, and in individual bundles of the frontalis and other facial muscles.

Dr. Spiller did not think the case was one of organic disease, and agreed with Dr. Mills' diagnosis. He called attention to a case of unilateral muscular twitching of the face he had reported. The man lived a long distance from Philadelphia, and was not seen until about a year after the first examination, at the later period the myokymia had developed into a convulsive tic.

Dr. Eshner stated that he had often noticed, in his own person, especially when much fatigued, a twitching of the muscles below the lower lip and above the eyebrow.

Dr. Sinkler thought that the case was clearly related to that form of muscular spasm of the orbicularis palpebrarum, associated with fatigue or neurasthenia, or with the excessive use of tobacco.

Dr. Mills, in closing the discussion, stated that he considered the case peculiar in that the myospasm was distributed over such scattered areas, not being limited to the brow or face. The condition was made worse by exertion, and came on while the patient was working and studying very hard. He advised physical and mental rest, combined with suitable tonics.

A New Reflex: Paradoxic Flexor Reflex.—Dr. Gordon described and demonstrated a new reflex which had the same diagnostic value as exaggerated knee-jerks and Babinski's sign. He found it always present in association with increased patellar tendon reflex. He examined thirty cases of various organic diseases of brain and cord, in which an involvement of the motor tract was diagnosed from the classical symptoms, In these cases the Babinski's toe phenomenon was present, or only slightly marked, or entirely absent. Curiously enough in the majority of his cases Dr. Gordon found a sort of antagonism between the paradoxic flexor reflex and Babinski's sign: as they showed a tendency to replace each other. The value of this new reflex is particularly appreciated in those obscure cases in which Babinski's is either absent or very slightly marked, and a diagnosis of an organic disease is in doubt. The author cited a case of trauma to the back, observed at the Jefferson Hospital, in which this reflex was present some time before Babinski's made its appearance, but the knee-jerk was markedly exaggerated. He also observed the reflex on the non-paralyzed side in hemiplegia, in which, as is known, there is almost always found an exaggerated knee-jerk, and in which the non-decussating homolateral tract is supposed to be involved.

The paradoxic flexor reflex is, therefore, a sign pointing to an involvement of the motor tract. For the purpose of control Dr. Gordon examined 200 normal individuals, also individuals with organic diseases of other portions of the cerebro-spinal system (not of the motor tract), also cases of paralysis agitans with increased knee-jerk, and in all the paradoxic reflex was absent. The reflex is elicited in the following manner: The patient is sitting with his feet (not legs) on a stool. The examiner, who is always on the outer side of the patient, places his thenar and hypothenar on the inner side of the tibia, and with the fingers exercises deep pressure on the calf muscles (the pressure must be deep in order to be transmitted to the flexor muscles); the great toe, or all the toes, then extend. It requires some practice to elicit this reflex, but it is easily and distinctly produced.

Dr. Pickett thought this reflex exceedingly interesting, but that it bore a marked relation to Oppenheim's reflex, brought out by stroking firmly along the inner border of the tibia.

Dr. McCarthy stated that Dr. Gordon's reflex impressed him as a new method of bringing out Oppenheim's reflex. He thought Dr. Gordon's method of eliciting the reflex was an improvement over Oppenheim's method.

Dr. Mills did not think the reflex was to be considered the same as Oppenheim's reflex. He thought that all of these reflexes—Oppenheim's, Babinski's and Gordon's—are significant of the fact that the nerves that supply the musculature of the toes will produce a reverse movement, and various means of producing the same movement may be obtained.

Dr. Spiller stated that he also had been thinking of the resemblance to Oppenheim's reflex. He suggested that it would be well to determine in what proportion of cases the extension of the toes, as obtained by Oppenheim's method, was associated with extension of the toes as obtained by Gordon's method.

Dr. Dercum thought this reflex different from a skin reflex, as it was produced by deep pressure of the muscles. It does not resemble Oppenheim's reflex, but is something different and entirely new. He thought that if we have here another reflex that will help to differentiate between functional and organic disease it will be very valuable.

Dr. McCarthy stated that Oppenheim's reflex is not a skin reflex, in as much as Oppenheim takes the blunt end of a percussion hammer and presses deeply in the muscle.

Dr. Gordon, in closing the discussion in regard to this reflex, stated that in making deep pressure, as he did in bringing out his reflex, he irritated the gastrocnemius and deep flexors, and instead of getting flexion he obtained extension. He did not think it had anything to do with Oppenheim's reflex. Neither did he think it had anything to do with Babinski's reflex, which was obtained by irritating the skin of the sole of the foot. By pressing upon the flexors he got extension, and he thought this was certainly a paradoxical reflex. He had examined thirty cases, in a number of which the Babinski sign was absent, while his own reflex was present. There is a certain practical value in this reflex, because when Babinski's sign is absent there is a doubt whether the disease is organic or functional, and in these cases he thought his reflex would aid in the diagnosis. He had always found it present in cases with exaggerated knee-jerks, and never found it in cases of tabes, or in normal individuals. In hysteria it was absent. In paralysis agitans it was absent. He stated that he had made a study of this reflex for some months, thinking it a modification of Babinski's or other reflexes, but upon careful study he found it different, as it has relation only to stimulation of the flexor muscles; the tibialis posticus and peronei have a function different from flexion or extension of the toes.

Neuro-fibrillary Changes.—Dr. Ludlum made a preliminary report on this subject.

Dr. Pickett thought that if the neuro-fibrils, as Apàthy believes, are the conducting element of the cell, in them we should look for changes in some forms of insanity; and in accordance with Dr. Mills' suggestion about two years ago, when Shaffer's Weigert studies of the paretic brain appeared, it would seem that this method should be useful in comparative studies of different parts of the cortex. If a difference were noted in the destruction of the fibrils in different parts, the consideration of a number of findings should prove of much value. He thought this method a distinct advance over the method of Nissl for the study of insanity.

A Case of Mixed Aphasia, with Hyperesthesia and Partial Hemianopsia in a Left Hemiplegic.—Dr. Mills and Dr. Weisenburg reported this case. Dr. Mills thought the case an interesting one on account of the left hemiplegia and the aphasia. Whether the patient was a right-handed man he thought was open to doubt. He believed the man was ambidextrous. He could use his left hand with a great deal of facility, and his right hand for writing. It is a well-known fact that some persons who are born left-handed may be taught to write with the right hand, although they perform other acts with the left hand.

Dr. Dercum thought the explanation was that the man was left-handed. He thought that the majority of ambidextrous people are really left-handed, and are merely educated to use the right hand.

Dr. Weisenburg thought the patient would be considered a right-handed individual.

Periscope

NEUROLOGISCHES CENTRALBLATT

(Vol. 23, 1904, No. 7, April 1.)

1. Concerning Virchow's Cretin Theory. W. **Weygandt.**
2. The Cessation of Severe Lancinating Pains in a Tabetic After Twenty-eight Injections of Antirabic Serum. L. **Stembo.**
3. A Contribution to Our Knowledge of Tabetic Arthropathy and Bone Disease. J. **Kollerits.**

1. *Cretin Theory.*—Continued article.

2. *Pains in Tabes.*—A tabetic who has had very severe lancinating pains for a long time was bitten by a dog. In the course of fourteen days he was given twenty-eight injections of antirabic serum. The patient, as a result, is now entirely free from these pains.

3. *Arthropathy in Tabes.*—According to Charcot tabetic arthropathies are due to a lesion in the trophic centers in the anterior horn of the spinal cord, these alterations being secondary to the changes in the bones and joints. Volkmann thought they were always traumatic in origin, while Rotter is of the opinion that they are a part of an arthritis deformans. The author examined the bones and joints of a tabetic and found the periosteum of the femur loosened opposite the insertion of the quadratus muscle, this being probably due to the irritation of that muscle. The loosened periosteum formed a lamella of bone, this causing a bone cavity, which was attached to the femur. The cavity was filled with a yellow serous fluid, the whole condition resembling a bone inflammation. The author argues that the loosening of the periosteum was the traumatic cause of the arthropathy.

(Vol. 23, 1904, No. 8, April 16.)

1. Concerning the Central Nervous System in Cretins. **Dr. Bayon.**
2. A Contribution to the Clinic of Tuberculous Meningitis. **Karl V. Wieg.**
3. Concerning Virchow's Cretin Theory. W. **Weygandt.**

1. *The Central Nervous System in Cretins.*—The author had the opportunity to examine microscopically two brains of undoubted cretins. In the first case, sections taken from different parts of the cortex showed marked alterations in the walls of the blood vessels, veins and capillaries, these resembling hyalin degeneration. There was a large number of amyloid corpuscles in each section. The nerve cells were not altered, except that there was marked pigmentation and the dendrites were plumper than normal.

In the second case the brain was placed in formalin one and one-half hours after death. The changes here were the counterpart of the alterations found in the first case. There were no amyloid corpuscles, except a very slight number in the optic nerves, and the nerve cells were entirely normal. The blood vessels were thin and not congested. These alterations represented changes found respectively in the senile and the infantile cretin.

2. *Tuberculous Meningitis.*—It is well known that the clinical picture of meningitis of tuberculous origin may vary, so that it is almost impossible to make a correct diagnosis. The author records three such interesting cases. In the first there was an alcoholic delirium throughout the disease, in the second the symptoms were ushered in by a violent delirium, and in his last case the clinical picture was that of an intoxication psychosis.

3. *Cretin Theory.*—Continued article.

(Vol. 23, 1904, No. 9, May 1.)

1. Several Theses Concerning the Order of the Motor Cells in the Origin of the Nerves of the Extremities. G. Bikeles.
2. A Contribution to the Innervation of the Eyes of the Mammalia. M. Bielchowsky and B. Pollack.
3. Concerning Virchow's Cretin Theory. W. Weygandt.

1. *Order of Motor Cells.*—(a) The ventromedial group consists, besides the large commissure cells, of the large motor cells for the musculature of the back, and for the innervation of the region of the ramus dorsalis posterior.

(b) All other cells, including the region of the ramus anterior, supply the ventral and the dorsal regions of the musculature of the extremities. These motor cells are arranged in the spinal cord according to the following morphological types:—

1. In a section of the spinal cord, studied sagitally, the cells in the anterior portion supply the musculature of the proximal distribution—as the muscles of the shoulder and buttock, and the dorsal cells supply the distal musculature.

2. In sections studied frontally the lateral cells correspond to the dorsal musculature, and the medial cells supply the ventral musculature.

(c) The different localization of these motor cells, whether dorsal or ventral, proximal or distal, are caused by the situation of the spinal nerves, and form a nervous localization in spite of the fact that the type is not strictly nervous.

Finally, the arrangement of the motor cells, after morphological principles, gives the best explanation for its correct functions. The anatomical representation of physiological complexes does not give satisfactory results.

2. *Innervation of the Eyes of Mammalia.*—The authors modify Bielchowsky's original silver impregnation method so as to be able to examine the eyes of mammalia. The article is not suitable for abstracting.

3. *Virchow's Cretin Theory.*—Weygandt discusses Virchow's "newborn" cretin theory, and presents from Virchow's many able works upon this subject the views held by that author. According to Virchow that in these cases there was a congenital disturbance of the synostoses of the cranial bones, Weygandt assumes to be incorrect. These "new-born" cases rather belong to that class of diseases which belong to the chrondrodystrophia fœtalis or mikromelia.

(Vol. 23, 1904, No. 10, May 16.)

1. Concerning a Peculiar Reflex in the Extremities in Central Organic Paralysis. W. W. Bechterew.
2. Concerning the Pathogenesis of Progressive Paralysis and a Contribution to the Anatomy of the Pyramidal tract. Dr. Bumke.
3. The Strength of the Tendon Reflexes and their Alteration in Hemiplegia. K. Pandy.

1. *New Reflexes.*—Bechterew recalls that in 1895 he described a phenomenon which was obtained in hemiplegics in the following manner: Both arms are flexed at the elbow, the muscles being absolutely relaxed, the paralyzed arm will not immediately extend, but there will be a momentary tension of the biceps, and then a gradual extension. Moler recently obtained this phenomenon in hemiplegies where there was no spasticity. Bechterew believes this to be an important sign, especially when hysterical hemiplegia is to be differentiated.

2. *Progressive Paralysis.*—Bumke recorded an interesting case of progressive paralysis of the insane in which there were Jacksonian convulsions limited to the right side of the body. At the autopsy nothing was found microscopically, except that the convolutions of the left side, especially in the Rolandic region, were somewhat flat and smaller than normal.

Microscopical examination by the Marchi method, a primary degeneration of the motor tract was traced from the left internal capsule to the lumbar cord. The cortical cells, especially the large pyramidal cells of the left central convolutions, were much altered. Nothing was found to explain the degeneration, so Bumke rightfully assumes that this degeneration was secondary to the alteration of the motor ganglion cells. There was marked arteriosclerosis. Jacksonian convulsions in this case cannot be easily explained. The especial involvement of the motor region in progressive paralysis is unusual, but has been previously noted. The cortico-bulbar fibers to the seventh and the twelfth nerves were traced. The degeneration in the foot of the cerebral peduncles was limited to the middle part. Bumke then discusses the probable course of the cortico-bulbar fibers, in this case it being in the medial instead of the middle part, and assumes, with Wiedersheim, that there may be two cortico-bulbar tracts, and these may not be diseased in the same individual.

3. *The Tendon Reflexes.*—Pandy constructed a mechanism by which he is able to measure the strength of the patellar reflex. He also discusses the various theories of the causation of exaggerated and diminished tendon reflexes. Acording to him this is clearly explained as follows: If the lesion in the spinal cord is of a non-irritative nature, whether acute or chronic, the reflexes will be absent. If the interruption is of an irritative nature the reflexes will be increased, as, for instance, the return of the tendon reflexes in a tabetic after an apoplectic attack. WEISENBURG.

JOURNAL DE NEUROLOGIE

(IX., 1904, 18.)

1. Remarks on Some Tics of Childhood. H. MEIGE and E. FEINDEL.

A discussion of the pathogenesis, symptoms and treatment of certain tics of children, which is illustrated by the histories of four patients. The authors consider the tics as beginning usually as bad habits, most commonly in children of neurotic constitution. They call attention particularly to tics of the eye muscles, precipitation in speaking, and in the act of eating. The treatment recommended is chiefly disciplinary, and what they call "psychomotor," a re-education of movements, by suitable exercises often before a mirror, with attention to the general health, and insistence upon proper rest, often a week or two in bed. In one of the cases mentioned there were the germs of certain obsessions, which the authors insist must always be looked for and nipped in the bud. The home relations of the child should also be inquired into, and regulated as far as possible.

(IX., 1904, No. 19.)

1. The Fundamental Principles of Electrotherapy in Nervous Diseases. E. DOUMER.

The author makes a plea for the more general use of electricity, which he thinks we have no more right to neglect on account of not understanding its mode of action, than we have to reject drugs of whose action we know little or no more. He maintains that electricity has a direct action upon the tissues outside of any chemical effects which it may set up, and cites some experiments with static electricity and with high-frequency currents to prove it. This action seems to be in the line of improving nutrition, and largely through its effect upon the vasomotor system, though he denies to electricity a specific action upon the nervous or muscular systems. The instances of its therapeutic action which he cites are mainly in the line of skin diseases. He claims to have cured eczemas and painful anal fissures by the action of the electric breeze, and by high-frequency currents. The more acute the condition the more rapidly it yields. He recommends the systematic use of electricity in wasting diseases, on account of its action in increasing the nutrition. The author does not particularize with regard to its therapeutic application in diseases of the nervous system, which he says depends upon the general principles which he has stated, except to advise beginning to use it earlier in acute conditions (as poliomyelitis and cerebral hemorrhage), at which time he thinks the probability of getting appreciable results much greater. The form in which it is applied, galvanism, faradism, static or high-frequency currents, he thinks is mainly a matter of convenience, as each exerts to a greater or less extent the specific electric action. Galvanism, however, is specially marked by chemical effects, which may be according to circumstances, desirable or undesirable.

(IX., 1904, No. 20.)

1. Neuraxology and Neuraxopathology. Heldenbergh.

Under the above title the author advances arguments for the study of psychology more strictly upon the basis of the normal and pathological anatomy of the nervous system. He lays special stress upon the relations of the cortico-pyramidal—or, as he prefers to call it, the psycho-cortico-pyramidal system, and the cortico-extrapyramidal system of fibers, the latter of which he regards as reflex and more or less involuntary, the former as voluntary and inhibitive in function. The study of the various mental and reflex phenomena, in the light of these relations, he suggests should form a new department, neuropsychology and neuropsychopathology, or shorter, "neuraxology" and "neuraxopathology."

2. Contribution to the Symptomatology of Nodding Spasm. Decroly.

The author reports the cases of four defective children of tender age who presented spasms of the above character, probably as an epileptic manifestation, and takes the occasion to discuss the nature of nodding spasm, about the relations of which there seems to be considerable confusion.

(IX., 1904, No. 21.)

1. Left Lateral Decubitus as a Means of Arresting the Epileptic Crisis. M. Lannois.

After hearing a communication, made by Crocq, at the recent Congress of Alienists and Neurologists of France, on the subject of his experience with this simple mode of treatment, proposed by McConaghy in the *British Medical Journal* for May 28, 1904, the author was led to try it in his epileptic wards. He did not have as brilliant results as Crocq, the manœuvre cutting short the paroxysm of epilepsy in only four out of twelve cases. He explains its action as due to interference with the return circulation from the brain by pressure upon the jugular vein of the opposite —the right—side, particularly through the tension of the omohyoid muscle, the brain in the first stage of the convulsion—the only time at which the method is useful—being anemic. Left lateral decubitus works better than right, since the right jugular vein is larger, and carries more blood than the left.

2. Traumatism and Alcoholic Delirium. H. Mabille.

A short discussion of the relation of trauma, both physical and moral, to the outbreak of delirium in alcoholic subjects, with reports of three illustrative cases. In many instances the delirium seems to be due to enforced abstinence.

Allen (Trenton.)

ALLGEMEINE ZEITSCHRIFT FUER PSYCHIATRIE

(Vol. 61, 1904, Pt. 5.)

1. On Ganser's Symptom. R. Henneberg.
2. Occurrence of General Paresis in Families. A. Marc.
3. A Case of Brain Abscess with Katatonic Symptoms. K. Schmidt.

4. Simultaneous Appearance of Mental Diseases in Two Brothers and a Sister. F. SKLAREK and C. F. VAN VLEUTEN.
5. Statistics in Asylum Reports. A. KOLLER.

1. *On Ganser's Symptom.*—The symptom of "talking around it" ("Danebenreden" or "Vorbeireden"), that is, of reaction upon the part of the patient to questions, by replies which, though incorrect and generally silly, still bear some relation to the subject, has formed the theme of a number of communications during the past few years, though its entry into the symptomatology of mental disease is to be credited to Ganser. The author, who has studied this symptom in a number of different cases coming under his observation at the Berlin Charité, comes to the following conclusions: In its typical form he does not find it very common. In the great majority of instances it was observed in hysterical cases—for which, indeed, it was considered as specially characteristic by Ganser—and of these a number were under criminal charges; others were affected in sequence to a trauma. It was also observed from time to time in cases of katatonia, in maniacal exaltation and delirium, and in simulation. On the whole the author cannot think it a symptom of any one form of insanity. Considering its psychology, he calls attention to the fact that traces of it are often found in the speech of mentally sound persons under circumstances of great fatigue, sleep drunkenness, and when replying to what appear to them as foolish questions, deserving a foolish reply. A healthy person also, when asked to simulate insanity, will often reply to similar questions, much as a patient with Ganser's symptom. The possibility of simulation, intentional or not, is also to be considered, and when the well-known susceptibility to suggestion of hysterics is remembered, that the questions intended to bring out this symptom are well calculated to exercise such a suggestive effect, is not surprising. A person accused of crime is often and naturally anxious to appear ill and mentally unbalanced, and here the suggestive effect of time, place and circumstance, together with suitable questions, becomes overpowering. On the whole the author sounds a note of warning as to the necessity for care in the questions intended to elicit this symptom and for caution in its interpretation.

2. *The Occurrence of General Paresis in Families.*—As showing that heredity may play a rôle in the etiology of general paresis, even in the absence of syphilis, the author gives the histories of three families. In the first of these (Langer) the grandfather, great uncle, father, uncle, aunt, the patient under observation (George L.), his sister, cousin and son were affected with general paresis. In the second (Bott) the grandfather, father, aunt and patient studied (Anna E. née B.) presented this disease. In the third family (Haaf) brother and sister developed general paresis. The parents were healthy, but a paternal uncle was "temporarily insane." It is not uncommon to find general paresis in families presenting also other types of mental disease. The author mentions several combinations illustrating this.

3. *A Case of Brain Abscess with Katatonic Symptoms.*—Case of a woman of twenty-six years of age, with strong hereditary predisposition to insanity on the maternal side, always peculiar and below the normal mentally, who gradually developed symptoms of katatonic character, and died after nearly three years' persistence of the mental disease under symptoms of exhaustion. At the autopsy an abscess, about the size of a small hen's egg, was found in the left parietal lobe, with a few nodules (tubercular?) in the lungs. The author weighs the symptoms in an attempt to decide whether or not the abscess might have existed since the beginning of the disease and have determined the katatonic manifestations, but feels unable to draw any positive conclusion.

4. *Simultaneous Appearance of Mental Disease in Two Brothers and a Sister.*—A contribution to the subject of communicated insanity. The

eldest brother, thirty-five years old, developed hallucinations of sight and hearing with delusions of being persecuted by a fellow employee. He spoke continually of persons entering his appartments, to rob and to abstract papers valuable to him, remained awake at night to watch, and armed himself against the invaders. His ideas became so impressed upon his brother and sister that they developed similar delusions about robbers, etc., and finally one night, all the parties being convinced that they heard (and in the case of the elder brother *saw*) the intruders, made a violent attack upon a fellow occupant of the house, who was descending the stairs upon his way to work. For this they were arrested and sent to Dalldorf for observation. The hallucinations and delusions persisted in the case of the elder brother, but the condition of the younger brother and sister speedily cleared up, leaving only the evidences of congenital mental deficiency.

5. *Statistics in Asylum Reports.*—A plea for the introduction into asylum reports of statistics of more practical character and resting upon better established facts than the customary ones bearing upon alleged causes of insanity, heredity, etc., not to speak of those setting forth the number of eggs laid by the hens, or quantity of hay cut upon the farm. The author thinks that more could be gained by careful figures as to number of patients isolated, number in prolonged bath, under hypnotics and other drug treatment, how many employed, etc., and the percentage relation of these to the whole number in the asylum. He gives some illustrative tables from Cery Asylum, Lausanne, and compares their figures with the corresponding ones from other asylums. ALLEN (Trenton.)

(Vol. 61, 1904, No. 6.)

1. The Biology of the Speech Apparatus. O. GROSS.
2. The Connection Between Aphasia and Insanity. O. ALBRECHT.

1. *The Biology of the Speech Apparatus.*—A study of the phenomena observed in aphasia, with an attempt to explain the connection of speech with other psychical functions upon biological and psychological grounds. The author pays special attention to the relation between gesture or sign language, and spoken and written language, and its impairment in cases of aphasia, four typical examples of this latter condition being described at length for this purpose. In the development of means of expressing wants and ideas, gestures undoubtedly were in earlier use than sounds, and they still play an important rôle in speech. The speech mechanism, receptive and emissive, is composed, not of a single sense apparatus, but of a synthesis of such apparatus, and in its function is closely connected the general mental processes, especially with the consciousness of one's own personality in relation to the outer world. Depending upon what part of this mechanism is disturbed, and the extent of such disturbance, compensation to a greater or less extent is possible. The author draws a distinction between physiological correction and biological correction, using the first term for the case in which the lost capability is again regained, the second for that in which the place of the lost capability is supplied to a greater or less extent by bringing into play another part of the speech mechanism, for instance, of conveying ideas by gesture language when the motor speech function is interfered with. Into the author's somewhat extended consideration of the psychological relations of speech it is impossible to enter here. He sums up in the following conclusions:—

1. "The sum of all the accomplishments of the organism which have relation to the common biological aim of understanding is a specific component of orientation, held together and regulated by a special system.

The ability to understand, to conceive and to reproduce explanatory actions as such, should be denominated signal orientation.

2. "The region of the brain called the speech field is to be considered as the center for signal orientation, and serves not alone audible speech, but accomplishes the binding together of all perceptive and reactive capabilities of all sense systems, in so far as these have relation to the single biological tendency to explanation.

3. "The common centralization of all possibilities of explanation causes the disturbance of gesture speech in aphasia from extended focal lesions.

4. "When in severe aphasias there are disturbances of gesture language, the disturbances of audible speech, and those of gesture language, appear as coördinated symptoms of focal disease.

5. "The more extended injuries of the center for signal orientation alter all psychical qualities, so far as these are related to the faculty of understanding.

6. "The self perception of aphasic symptoms is dependent especially upon the conceptibility of the components of the understanding.

7. "It is possible that the center for signal orientation is to be considered as the cortical representation of a subcortical apparatus, from which the impulse for the automatic actions of imitation of stimuli is set off, and that it may be the function of the cortical center through its intimate relations with the rest of the cortex to differentiate from these automatic actions signal orientation as a component of general orientation."

2. *The Connection Between Aphasia and Insanity.*—Aphasia and insanity may be combined in three ways: 1. Both are symptoms of a common cause. 2. The aphasia is a result of the mental disease. 3. The psychosis is a result of the preceding aphasia. The author discusses these relations at length, and gives full histories of two cases, illustrating the third manner of relationship. In each of these cases (both males and alcoholic) an apoplectic stroke had, as a result, the production of aphasia of mixed character, but chiefly sensory, to which hallucinations of sight and hearing, and possibly also of taste and smell, were added after its persistence for a considerable time, with the development of ideas of persecution, leading to the production of a paranoid state with increasing weak-mindedness. The author, after studying these cases, and reviewing the literature of the subject at some length, comes to the conclusion that this paranoid state originated psychologically from the change in consciousness of self by reason of losing touch with the outer world on account of loss of ability to understand and to express himself on the part of the patient, and might have as its anatomical basis changes proceeding secondarily from the focal lesion, or possibly from some obscure toxic influence connected with neurone degeneration. Allen (Trenton.)

JOURNAL OF MENTAL SCIENCE

(Vol. L., 1904, July.)

1. Quantitative and Qualitative Leucocyte Counts in Various Forms of Mental Disease. Bruce and Peebles.
2. Notes on Social Causes of Alcoholism. Sullivan.
3. General Paralysis and Crime. Baker.
4. Acute Hallucinatory Insanity of Traumatic Origin. Drapes.

1. *Leucocyte Counts in Mental Disease.*—This article embodies the result of careful and laborious and extended investigations, and appears to us to be most trustworthy. They take as a normal percentage in control the following: Polymorphonuclear, men, 70 per cent.; women, 60 per cent. Small lymphocytes, men, 20.5 per cent.; women, 30.5 per cent. Large lymphocytes, 8 per cent.; women, 7.5 per cent. Eosinophiles, 1.5 per cent.; women, 2.0 per cent. Mastzellen, .5 per cent. to 1 per cent. Acute melan-

cholia—Here there was never a leucocytosis. If in an early stage of melancholia a hyperleucocytosis was observed they made a guarded prognosis. Excited melancholia—When examined early usually a high count was obtained. Polynuclear above 70 per cent.; leucocytes 11,000 to 17,000. Later on this would be respectively 80 per cent. and 20,000 to 30,000. The onset of a relapse would often be preceded by a fall to, or below the normal. With the increase in excitement there is an increased leucocytosis, as also percentage of the polynuclears. Acute continuous mania—The leucocytosis resembles that of excited melancholia, the hyperleucocytosis often persists after recovery. In cases which do not recover and become chronic the leucocytosis falls, and the percentage of polynuclears also to 50 per cent. or less. If the chronic cases eventually should recover there is a rise in both the above factors. A point of practical importance is here indicated for a low count of whites less than 10,000, and a low per cent. of the polynuclears, means a bad prognosis. The leucocytosis may be artificially raised by injecting ½ c.c. of turpentine into the flanks; this procedure has also been followed by rapid recovery. Recurring mental excitement—Folie circulaire. All the cases exhibited depression or excitement of the simple mania or melancholia forms. In the depressed, invariably, high leucocytosis, polynuclears 60-80 per cent. During beginning of excitement, fall of leucocytes, polynuclears also, to about 50 per cent. As the excitement increases a rise in both these factors was observed. Two typical cases were discharged with very high leucocytosis and high per cent of polynuclears. These observations point strongly to the fact that the depression and excitement of cases of "folie circulaire" are quite different from ordinary attacks of mania and melancholia. Cases of recurrent mania (exclusive of the alcoholic cases)—Here there is a rise of both leucocytosis and per cent. of polynuclears. The rise occurs gradually as the attack supervenes, and falls slightly as it passes off. No per cent. of polynuclears follows the general curve of the hyperleucocytosis. One of their cases suffered from severe intercurrent facial erysipelas. It showed hyperleucocytosis 28,000; per cent. of polynuclears 89. Following this illness there was a cessation of mental attacks for four months. Alcoholic insanity. No case of delirium tremens was examined, but the leucocytosis of acute continuous mania (alcoholic origin) was identical in every detail with the non-alcoholic variety. They look upon the alcohol merely as an exciting cause, breaking down the resisting power of a patient hereditarily predisposed to this type of insanity. Hebephrenia—The findings here were variable, for the most part leucocytosis 12,000 to 14,000, but often marked hyperleucocytosis, without increase of polynuclears, but an increase of large manonuclears. Only one case recovered—here the leucocytosis was never above 14,000, and fell to normal on recovery. Delusional insanity—These cases, whether of paranoia or occurring later in life, were always entirely free from hyperleucocytosis. Progressive paralysis—At the onset there was a high per cent. of polynuclears and hyperleucocytosis. During the second stage, especially if there was a febrile attack, there would be a rise. In the third stage there was generally a variable leucocytosis, 30,000 to 10,000, but usually a low per cent. of polynuclears—40 per cent. If recovery or marked remission ensued there was a return to normal figures. Epilepsy—In every case, either during or between attacks, has shown a hyperleucocytosis. Puerperal insanity—The leucocytosis does not differ from that of acute mania. Lactational insanity—shows conditions as in ordinary anemic women. Conclusions—With the exception of one variety of melancholia and all cases of delusional insanity, all types of acute mental disease present more or less the conditions of hyperleucocytosis, with persistance after recovery. Difference in the character of the corpuscles is very marked, they being large and well formed in young, strong patients, smaller and staining badly in the old or debilitated. Large lymphocytes (uncommon in healthy blood) are frequent in various forms of mental disease, but most markedly found in hebephrenia, catatonia, recurrent mania and in any case which is much debilitated.

2. *Alcoholism.*—We cannot better review this very able and interesting article than to give the writer's summary in his own words: 1. In considering the social causes of intemperance, which are by far the most important, it is necessary to distinguish two opposed types of drinking, viz.: the drinking that goes with conditions of relaxative luxury, and finds its most frequent expression in ordinary convivial drinking, and the drinking that goes with conditions of relative misery, of which bad industrial circumstances are the most considerable factor. 2. Convivial drinking may, and often does lead to drunkenness, but, at least in its pure form, does not tend very much to chronic alcoholism. Industrial drinking, on the other hand. while leading also, though less immediately, to drunkenness, tends rapidly and fatally to chronic intoxication. 3. From a statistical point of view, while chronic alcoholism always implies the existence of drunkenness, drunkenness by no means implies the existence of chronic alcoholism. In England this divergence between the two phenomena is best seen in the prosperous mining districts, which by reason of their prosperity are at once more drunken, but less alcoholic, than any other part of the country. 4. The graver social evils that are in any important degree caused by alcohol are related to the chronic intoxication, and are, therefore, mainly due to industrial drinking. 5. While educational, religious and similar influences can control the excesses of convivial drinking, they have but little action on industrial alcoholism, which can only be checked by raising the standing of living, and, in a minor degree, by such methods as restricting the facilities for obtaining alcohol during work hours, providing hygienic substitutes, and so forth.

3. *General Paralysis and Crime.*—There is a small percentage of cases where general paralytics show criminal tendencies. Some excellently recorded cases are cited to show this relationship. In such cases the crimes were committed early in the onset of the disease before the somatic symptoms had presented. The crime was associated with and was the outcome of melancholic conditions or states, and was homicidal in nature. The tendency to suicide is not marked, being probably inconsistent with the optimism and egotism so characteristic of the general paralytic. Delusions of persecution were always present in these cases. This invasion period of melancholia and persecutory mental disorder Baker views as the initial stage of a later dementia, and considers them sufficiently fixed and systematized—not fickle and inconstant—to cause resolute and purposive homicidal acts. The victims were usually the offspring of the destroyer. Baker objects to the phrase "homicidal impulse," as applied to criminal acts, impulse meaning mental force suddenly liberated, and as suddenly transformed into action. There is nothing sudden about these acts of violence. The thought of violence when it projects itself into the mind rarely leads to immediate action, but grows with the growth of insanity, and requires time for development to its full intensity. They are, indeed, usually foreseen and feared by the patient, indeed, fill him with horror, and there is a constant struggle between them and the remnant of patient's weakened volition. The affective state in such patients is usually a very gloomy one. Sexual offences Baker attributes to stimulation of the genital sense by alcohol, this agent playing always the principal part. He quotes illustrative cases where sexual offences were committed and conviction followed for crime which were later followed by typical dementia. Offences against property: The thieving propensities of the paralytics may be ascribed in great measure to reversion by a process of dissolution, to conditions similar to those of childhood, wherein the acquisitive tendencies form a prominent feature of mental activity. The instinct of acquisition is uppermost, and is not yet held in check by those higher inhibitory intellectual and volitional processes which develop later and fit the individual

into our social sphere. In the paralytic this cortical superstructure melts away under the influence of the destructive process and the inverse order of acquirement; the highest and latest developed disappearing first. Baker, in fact, thinks that general paralysis gives a replica of childhood not paralleled in any other form of insanity.

4. *Hallucinatory Insanity.*—Drapes gives an extended description of a case. It is remarkable for the evidently established connection with the trauma, and the connected character of the hallucination. They were extremely vivid and coherent and made such a strong impression that they remained fresh in the patient's memory five months after. They were as real as dreams, but occurred whilst patient was broadly awake, and again were different, in that dreams never, or rarely, incite to action, whilst here striking displays of motorial activity occurred on several occasions. Interesting, as furnishing ground for favorable prognosis, was the fact that a lucid interval was observed every day, replacing the periods of hallucinatory excitement. Drapes conjectures as to the nature of the lesion which could cause such a peculiar derangement. He excludes concussion, of which he had thought at first. There were present gravescent headache, with a certain amount of hyperacusis (this, too, in a patient who had always been slightly deaf), slight paretic phenomena early, viz.: drooping of eyelid and face and slight dragging of leg, muscular twitchings, irregular pulse and insomnia. The primary physical symptoms were followed about two to three weeks later by the mental; hence, Drapes pictures the lesion as a circumscribed meningeal inflammation which later involved, but not very deeply, the underlying cortex. Drapes further discusses most interestingly the bearing of his case upon the nature of consciousness, of personality. Human consciousness formerly was considered as a sort of essence single and individual pervading our whole being. Modern psychology postulates consciousness, the ego or personality as the sum of a vast number of separate consciousnesses, the sum of which is general consciousness. This consciousness is a reaction to outward impressions, is the outcome of objective and subjective consciousness, and instead of being changeless and undesirable is in reality never the same for any two consecutive periods of time. Each new phase of consciousness awakens memories of innumerable past states, and the new phase, more or less vivid, tends by repetition to become automatic or reflex. Drapes believes in what has been termed the stratification of the mental faculties, and with various levels are associated corresponding states of consciousness, *i. e.*, personality. The highest level holds in control subordinate or lower levels of consciousness. These conscious states become the dominant states when higher levels are rendered inoperative by lesions of various nature. There may be any number of different personalities, and for every stage of descent in the process of dissolution there is a different consciousness or personality. Sleep, healthy and dreamless sleep, is defined as sopor in all levels ordinarily attended with consciousness. "Le sommeil géneral est l'ensemble des sommeils particuliers." Dreams are the persistence of activity in one or more levels whilst the others are temporarily inactive, due to the activity of subordinate centers, relieved from the control of those above them. Hallucinations in Drapes's case were explained by the same hypothesis. The higher centers were rendered inoperative by the circumscribed meningitis, and the lower ones "let go." The vividness of the hallucinations were explained by the supposed irritative effect of the neighboring lesions. Drapes's idea of the origin of the delusions of persecution, plotting, conspiracy against life or property so common in all forms of insanity is ingenious. The mental ground work common to the race is much the same in all beings—insanity being merely a reduction from a higher to a lower level of consciousness—degradation of personality. The basic state of these delusions is fear and suspicion, dating back from long forgotten but inherited ancestral experiences accompanying human evolution. With the extinction of activity

of the higher, but more recently evolved and super-imposed cortical levels, there will be a revivification of these more primordial states. which in proportion as the control of the upper levels is removed, permits them to emerge into the foreground from the depths of our psychic life.

Wolfstein (Cincinnati.)

ARCHIVES DE NEUROLOGIE

(Vol. XVIII., 1904, No. 105, September.)

1. Frequency and Distribution of Naevi Among the Insane. Féré and Moroux.
2. Phobia of Inspection. Hartenberg.
3. Dementia Præcox. Fourteenth Congress of Alienists and Neurologists of France and French Possessions, Pau, August, 1904.

1. *Naevi.*—This article discusses the condition named in the title, and is a study of the various forms as they occur in the various types of the insane. The conclusions at which they arrive show similar conditions here to those met with in sane subjects. They do not support the theory of Bouchard, that there is any connection between the occurrence of these cutaneous affections and diseases of the liver.

2. *Phobia of Inspection.*—In this contribution Hartenberg describes the phobia of inspection, especially that form which results in aversion to the exposure of the face to the public view. He describes the emotional excitement and unexplainable sensations always resulting when the sufferers have to support the fixed gaze of others. The natural consequence of this fear is to avoid as much as possible the necessity of exposing the face. Either these occasions are avoided altogether or all kinds of expedients are adopted to avoid inspection; such as covering the face with a veil, or with the hands, or standing in the shadows, or if these cannot be adopted the face is held expressionless, or some artificial expression of the countenance, or even tics are adopted. Others again will always wear blue glasses. *The basis of this phobia is timidity,* and these subjects always present the symptoms of this social infirmity, which probably arises from the fear of being found mal-formed or ridiculous, and results in an excess of *shame.* When it is confined merely to fear of inspection of the face, or even the eyes alone, Hartenberg thinks it is rather because they feel that inspection of these parts is a sort of profanation, constituting a violation of their conscience. He contrasts this phobia sharply with the phobia of blushing. The victims of this inspection phobia never blush, or if they do, are unconcerned about the blushing. It is merely the exposition of the naked face, or of the eyes, to others, which is the cause of their disquietude. The history of two very interesting cases is given, which Hartenberg considers very critical examples, of the malady. Summarizing the condition, this phobia is a morbid fear ingrafted on the mental state of timidity, similar to the phobia of blushing, but entirely distinct from the latter. It is a particular form of the class of social phobias.

3. *Dementia Præcox.*—The proceedings of the Congress were extremely interesting, but it is not possible to report on all of them in detail. The subject of Dementia Præcox was the special topic for discussion, and the disease as described by Kraepelin was presented to the Congress by Deny. Kraepelin's views were ably defended, and Deny believes that it is as distinct a form of dementia as is that of general paralysis, or senile dementia, and he considers it a morbid entity. The intellectual deficit is first manifested in an elective manner, affecting the emotional and moral side, rather than the voluntary activity, or higher intellectual faculties proper. Apathy and indifference are its earmarks; later come disorder of volition and motorial activity. Characteristic is the progressive weakening of the intellectual faculties to which is usually joined states of depression, or excitement, or confused stupor, or delirious con-

ceptions. This intellectual reduction in dementia præcox has characteristics which clearly distinguish it from the other well defined dementias. *Automatic motor activity* is nearly always preserved, in fact, even exaggerated; *the power to arrest it is weakened or lost.* The combination of diminished voluntary activity and uncontrolled automatic activity is the basis of the syndrome catatonique. There is either negativism, with a negative activity, or manifestations of the suggestible state may take the form of undue imitative activity (catalepsy, echolalia, echomimia, echopraxia). These two groups of phenomena are in affinity, and may co-exist alternately. They are underlaid by an identical psychic process, namely, disorder of voluntary activity, with simultaneous persistence of a certain unregulated automatic activity. The modifications in the intellectual sphere are shown by lack of attention, loss of memory, of reflection, judgment and association of ideas. The foundation stone of the malady is this primary intellectual enfeeblement. Without hallucination, without excitement or depression, this intellectual reduction constitutes the simple type of dementia præcox (forme simple—Serieux). Hebephrenia, catatonia, or paranoid states occurring in connection with the dementia præcox, are super-imposed conditions. Deny thinks the delire polymorphe des degeneres, the delire d'emblé of the French, the acute paranoia of the Germans are closely allied, in fact, should be considered as identical with the varied systematic delusions and hallucinations of dementia præcox. In catatonia especially, do we see these persistent types of systematic delusions. When interrogated during remissions, or lucid intervals, as to their reasons for their negativism, stereotypy, or imitation, the patients either gave insignificant answers or they denied the possession of all *desire* or *volition.* Deny agrees with Seglas that the only plausible explanation of the syndrome catatonique is to be sought in the passivity of the mind and slowness of the psychic processes, and the progressive mental reduction.

Physical Signs.—These are not well defined. Nearly all writers on dementia præcox mention exaggeration of the tendon reflexes, especially in states of stupor or semi-stupor. On the other hand, in many cases the cutaneous reflexes are abolished. The pupils show greater variation in the light, the accandation reflexes as well as occasional change of contour. Cyanosis and coldness of the extremities, localized edemas, pseudo-edemas, alternative emaciation and obesity, coming on rapidly without appreciable cause (Seglas)—dermographism—are all mentioned. The semeiological value of the physical signs is not great. Deny thinks they are more marked and numerous than in other psychoses, and when taken in their ensemble they may contribute somewhat towards a diagnosis. Deny now discusses the justification for the nosological anatomy of dementia præcox; its symptomatological anatomy being well defined. Is dementia præcox an accidental or constitutional psychosis? Viewed from the standpoint of causation two factors are in sharp outline. 1. Is the existence of the majority of cases of unquestioned hereditary taint—neuro-psychopathic ancestors? That there is strong probability of an autotoxic process being the responsible causative element. This auto-intoxication arises either from disturbance of the sexual glands or some functional insufficiency of the ductless glands. Whilst conceding the rôle of heredity and pre-disposition, Deny has nothing better than generalization to offer for the theory of auto-intoxication. Deny, himself, is disappointed in his efforts to support the nosological autonomy of dementia præcox on etiologic evidence, and appeals to pathology for assistance. Klippel and Lehrmitte have shown profound alterations of the cortical ganglion cells and their prolongations, and although these changes occur in other dementias and psychoses, Deny maintains, justly, that it is just in this cortical terrain, wherein the toxic processes must exert their power for deleterious action. Klippel and in the course of the periodic melancholia of folie circulaire, or even in alcoholic melancholia; most often this symptom complex is observed in recurrent melancholia, with psychic obsessive states. (2) In these cases one may establish the presence of the caractere scrupulo-inquiet as one of the forms (the simplest) of the ideo-obsessive constitution. (3) Often in the coexistence of the melancholia state with the contrasted psychic obsessive associations the clearness and the intensity of the obsessive representations can appear in such degree that the latter, already invested with a sensorial coloring, may lead to the formation of a hallucinatory and illustory form of obsession.

2. *Anesthesia with Dreams.*—In an hysterical patient, *totally anesthetic,* the moment the eyes were closed during sleep, or when closed by the observer, all sorts of dreams, especially those of frightful character, accompanied by sensations of danger and personal damage, would occur, and great sense of fear would ensue. This would disappear as soon as the eyes would open. The author does not consider the condition due to somnambulism or to hypnosis, but thinks there is a direct connection between this dreamy state and the anesthesia. The patient was a total anesthetic, all external sensation was lost, and she was forced to rely upon *sight,* the only sense left, for information from without. She had no notion of time or space, except through sight; she lived by means of this one sense. The patient said, as soon as her eyes were closed, "I no longer see; I do not know where I am; I am suffocating; I am dying." With closed eyes she is incapable of a thought, of a conscious movement; she can hear, and move her members, and can raise herself from the bed, but unconsciously it would appear. One might say all connections cerebro-bulbar and cerebro-spinal are abolished. She is reduced to the elementary reflex state, and it is because she has no other perception of consciousness, be it external or internal, that she knows not what she does, that she believes she no longer exists. The dreams are the result of this absence of perception. In the normal state the sensation of well-being is determined by the equilibrium, the harmony of organic functions; destroy this, and two conditions present: either the subject is awake and then has a clear perception, consciousness of the anomaly of function, he suffers and can localize his suffering; or he sleeps and then can have only an obscure perception, sub-conscious, of his suffering; associations more or less bizarre form themselves, and the result is a nightmare, more or less violent. In this concrete case the patient was not asleep, but there is no way to differentiate this pseudo-wakefulness from pseudo-sleep, except that in the one case the eyes are shut, in the other open. Being an anesthetic, however, she had in neither case any clear perceptions. This state is neither wakefulness nor sleep, properly speaking. There is suspended activity of the cortical centers. This is the state which Sollier has called *vigilambulisme,* thus differentiating it both from normal sleep and from hypnotic sleep. In this case all the nightmares were built around the one sensation, viz.: the fear of falling, the fear of suffcation. The patient having lost her sensation, cutaneous, muscular and osseous, to the extent that no peripheral impressions were received; not seeing her body she felt this sensation of falling (de chute) which everyone has experienced. No visceral sensations coming from the lungs, not feeling the air enter, she believed she would suffocate; as one does not suffocate or fall without reason, she associated these sensations, which (to speak correctly) were not sensations, but the interpretations of absent sensations, which should normally exist, with the ordinary causes of falling or suffocation; as precipices, falling walls, phantoms, which strangled her, etc. With these came terror, all the more intense to the patient on account of her general anesthesia, which rendered her incapable of escaping from the causes of death. She could not escape without opening her eyes, then she had visual proof that it was the sport of a dream, but she did not feel

it, but established *by vision* the absence of the causes which had worried her, and with these disappeared all the subjective conceptions to which these had given place. Why could not the patient spontaneously open her eyes? This the author also explains, not satisfactorily, on the basis of anesthesia and loss of muscular sense. The author thinks his case, when analyzed, shows a state of psychic and somatic deficiency not to be associated with either normal or hypnotic sleep, but to a differentiated state, called vigilambulisme by Sollier. Anesthesia is a primordial phenomenon of hysteria · this it is which dominates all the phenomena of this neurosis. The more complete the anesthesia the more pronounced were these nightmares. Indeed, in a foot note, the writer shows that there has been marked amelioration. The writer's summary is this, that the condition described in this case warrants the conclusion that the phenomena exhibited were the manifestations of a psychic state, created of one piece by the anesthesia.

WOLFSTEIN (Cincinnati.)

MISCELLANY

RESEARCHES ON THE MOTOR LOCALIZATION IN THE SPINAL CORD. G. Marinesco (La Semaine Médical, July 20, 1904).

The author makes an exhaustive study of spinal centers, with the conclusion that muscles, which possess a common function, are represented by cellular groups, which from an anatomical point of view are centered in a common mass, which experimentation may demonstrate. These centers are superimposed, arranged in the same order as the corresponding muscles. There exists, therefore, in the cord a veritable muscular projection, arranged according to the laws of organic symmetry. The functional groups are combined in a manner which the economy of material and space calls for, and as a result making possible a rapid and useful function.

(W. B. NOYES.)

A CASE OF DISLOCATION OF THE ATLAS. James Hendrie Lloyd (The Amer. Jour. of the Med. Sc., Nov., 1904).

The author reports a case of dislocation of the vertebræ similar to two previous cases reported by him in the JOURNAL OF NERVOUS AND MENTAL DISEASE of February, 1900. The patient, a man, twenty-one years of age, fell from a cart while drunk, striking his left shoulder, the cart wheel passing over the back of his neck. After that time he was unable to turn the head without at the same time rotating the shoulders. Four months later he had another severe fall, striking the back of his head on the pavement. This rendered him unconscious for five minutes, and when he regained consciousness he was unable to move any of his limbs. This paralysis lasted about an half-hour, after which he slowly regained power, first in the legs, then in the right arm, but none returned in the left arm. The patient had a marked deformity in the neck. The head was twisted to one side and slightly rotated, and with the finger on the pharynx the deformity could be felt as a projecting mass on the posterior wall. There was a paralysis of the left arm, not, however, complete, but none of the legs and no involvement of the bladder or rectum. There was a loss of pain and thermic sense on the right side of the body, more marked in the arm and forearm, and also an area of tactile anesthesia in the region of the distribution of the great occipital nerve of the left side. The skiagram shows a displacement of the atlas.

C. D. CAMP.

EXOPHTHALMIC GOITRE, WITH THE REPORT OF A VERY RARE CASE. J. W. Wright (The Columbus Medical Journal, Nov., 1904).

Patient, sixteen years old. At eight years of age protrusion of left eye with nystagmus of both eyes. Defective vision, left eye worse. Three years ago, total blindness of the left eye. Present time, protrusion of both eyes; left worse. Thyroid gland somewhat enlarged. Interesting features of case are age and loss of vision. Cause of blindness due to pressure of compact cellular tissue of orbit upon optic nerve.

J. E. CLARK (New York).

TRAUMATIC HEMORRHAGE OVER THE THIRD ANTERIOR FRONTAL CONVOLUTION; OPERATION; REMOVAL; RECOVERY; REMARKS ON PRESENT STATUS OF BRAIN SURGERY. William C. Krauss (Amer. Jour. of the Med. Sc., Sept., 1904).

The patient received the injury from a fall, striking the right side of the head on the pavement. He was not immediately unconscious; and the next day was, with the exception of bruises, apparently well. Symptoms of serious injury appeared gradually and progressively; headache, vomiting, aphasia and paraphasia, and on the fourth day Jacksonian epilepsy, involving only the right side of the face. A diagnosis of hemorrhage over the left frontal region from *contre-coup* was considered likely, and an operation performed. Three teaspoonfuls of blood clot was removed. Recovery was complete in one month.

C. D. CAMP (Philadelphia).

APHASIA AND THE CEREBRAL ZONE OF SPEECH. Charles K. Mills (Amer. Jour. of the Med. Sc., Sept., 1904).

Dr. Mills presents a series of personal observations demonstrating the existence of the centers of the zone of language. According to this exposition, the auditive center for speech is situated at the junction of the first and second temporal gyres, about opposite the point where the horizontal branch of the Sylvian fissure turns upward posteriorly; the dynamic, or motor speech center, occupies the hinder portion of the subfrontal or third frontal convolution, and, according to the views of the writer, the insula, or, at least, its anterior half; the visual centers immediately concerned with speech are located in the angular gyre; the site of the graphic, or uniting center, is in the caudal half or third of the second frontal convolution, and the naming center is probably situated in the third temporal convolution. These different centers are connected each to the other by a system of to and fro association, so that the impulses from special areas probably pass in both directions. Personal clinico-pathological cases, with necropsies, are given to show the correlations of the zone of language with the motor and sensory spheres of the brain, and also there is adduced the biological principle which is fundamental to our conception of the speech zone and its subdivisions, *i. e.*, that the centers for word-hearing, word-seeing, speech-associations and for writing have developed in the race and in the individual as these functions, or attributes have been added to man's resources for communicating by language with his fellows.

C. D. CAMP (Philadelphia).

THE TREATMENT OF CHRONIC INTERNAL HYDROCEPHALUS BY AUTO DRAINAGE. Alfred S. Taylor (The Amer. Jour. of the Med. Sc., Aug., 1904).

After a review of the literature of the subject the author relates the histories of six cases of operation for the cure of this condition with a mortality of 50 per cent. In only two of the cases was the disease process apparently arrested, and from his experience he draws the conclusion that the only feasible method of treatment is some form of internal or auto-drainage, which must be slow and either permanent or much prolonged; and also that in order to be successful the operation must be brief, must involve the escape of but a small amount of cerebro-spinal fluid, and should be performed early in the disease, before irreparable damage has been done to the brain structure. C. D. CAMP (Philadelphia).

Book Reviews

Clinical Lectures on Mental Diseases. By T. S. Clouston, M.D., Edinburgh, F.R.C.P.E., President of the Royal College of Physicians of Edinburgh, Physician-Superintendent of the Royal Edinburgh Asylum for the Insane, Lecturer on Mental Diseases in the University of Edinburgh. Sixth Edition.

Clouston might be called the "Grand Old Man" of psychiatry. His book has been standard for a generation, and remains one of the indispensable books to the alienist; for, however "advanced" the reader may be in pursuit of German thought he turns often to the clear, sober but vigorous descriptions of the Scottish Nestor. To one who has been (in the warning phrase of Dercum) "Kraepelin-getrunken," or to one who turns from Church and Peterson's latest edition after a perusal of Adolph Meyer's scholarly review of modern German psychiatry, how very salutary to listen again to the spokesman of the Royal Edinburgh Asylum, where every excited patient is labelled "mania" on admission, and every depressed one "melancholia"! At any rate it is restful. We are all liable to follow after taking terms in science; but Clouston's fault is perhaps the reverse; he disdains psychiatric technicalities. This is well illustrated on page 673, where Clouston speaks of post-operative insanity as "melancholia," though in the same paragraph he says his were all cases of delirium. If the reader will substitute "excitement" for mania and "depression" for melancholia throughout Clouston's book he will find it less crude and much finer in distinctions than he may have thought. Clouston paints from nature: the panorama of mental disease is in his book, and as he says of another (page 304), "all must agree with him for he sticks closely to clinical fact." His conception of circular insanity is restricted, but truer than that of the French writers; his states of defective inhibition embody a rational idea of morbid impulse, which some German writers have split and pulled to pieces and refined until it is gaseous; his phthisical insanity and some other symptomatic types are unsurpassed. The fault of Clouston's book is the fault of a colonial map of America; the original States are shown mainly as they are now and the whole country in outline; but the new boundary lines, and the territory since explored and divided into separate States, are not represented. In these "realms of gold" Clouston has traveled, but (carrying our simile farther) to him they are still a "western reserve" connected with the old States. In this edition Clouston draws upon Defendorf's translation to present, briefly, Kraepelin's views on dementia præcox; and he thus falls into errors of quotation; thus paranoid dementia præcox is characterized by—"persistence of delusions—in spite of progressing mental deterioration." The German word is ***neben***, "together with" symptoms of a rapidly-developing mental weakness. To point this out would be captious were it not that the great Scotch alienist is passing judgment on the German, and condemns him upon such evidence. The conception of dementia præcox has been expanded by Kraepelin, but the name as well as the conception has been slowly evolved. Even Benjamin Rush, in his "manalgia," presaged the dementia præcox of Kraepelin; and the strange genius of Edgar Allen Poe portrayed some features of it in his tale, "Bérénice." The psychiatric world to-day is making much of dementia præcox—perhaps too much, but surely it is a more definite and important thing than Clouston's Insanity of Puberty and Adolescence. Clouston is not a precisianist, as witness, his use of the terms delusion and sense-delusion for illusion (pages 210 and 250); this is darkening counsel in spite of Sully's use of the word illusion (page 163). Are not Clouston's scientific offspring in Edinburgh shocked by a diagnosis-like "hysteria, brain softening and spinal disease" (page 374), or by the idea of tabetic paresis "spreading upwards by a pathological propagation along the connecting nerves in the lines of physiological function till it reaches the brain-cortex"? Is it not the case that "ascending paresis" occurs usually in ordinary lumbo-thoracic tabes? Why does Clouston so vehemently deny the inflammatory origin of pachymeningitis interna? He doth protest too much; that question is not settled, and the trend of opinion is away from his view. Clouston's recognition of non-delusional paresis (in one-third of the cases, pages 392 and 393) in accordance with the views of Mendel and others,[1] and should be a hint to such authors as M. Allen Starr. Those who follow Clouston's advice (page 396) not to diagnosticate general paralysis from mental symptoms alone, will paradoxically find more cases in their wards than if they make the delusion of grandeur their Shibboleth. Clouston maintains (page 161), as he has from the first, that grave delirium is only an intense mania, and in this he has had the support of Meynert, who regarded it as the "highest intensity of confusion," and of Kraepelin, Wernicke, etc., to whom it is merely a severe grade of any one of several psychoses. After all, the main criticism of Clouston is but criticism of a school of thought in which he long has dominated. His plea, and every psychiater's, must be that of Paul—"I count not myself to have apprehended, but this one thing I do, I press toward the mark"; and by his zeal and strength Clouston has been for nearly half a century one of the foremost contestants for the prize of our high calling. WILLIAM PICKETT.

TRAITÉ DE MÉDECINE. Deuxieme Edition. Par M. M. BOUCHARD ET BRISSAUD. Tome IX. Par M. M. BRISSAUD. O. CROUZON, GEORGES GUILLAIN. G. GUINON, LAMY, A. LERI, P. LONDE, P. MARIE, A. DOQUES, TALLEMER, Diseases of the Nervous System. Masson et Cie, Paris. G. Stechert, New York.

It is now hardly more than two years since the first edition of this splendid system of medicine of Bouchard and Brissaud's was published In the present edition Vols. IX. and X. are devoted to diseases of the nervous system. In Vol. IX., before us, a very great revision has taken place, making the volume an entirely new one. Professor Brissaud contributes the section on Diseases of the Cerebral Hemispheres. It is a remarkable summary of this field of rapidly increasing facts. It is a monograph of 346 pages, the sections on cerebral localization, hemiplegia and aphasia being the most complete and clarifying that we have been privileged to read. Diseases of the Cerebellum are discussed by Louis Tollemer, and constitute a very great advance on most contemporaneous treatments of diseases in this region. The Cerebral Peduncles, the Corpora Quadrigemnia, the Medulla and their diseases are written by Georges Guillain, whose brilliant work has stamped this young neurologist as one of the leaders in the French Capital. With Pierre Marie, his chief, he has written the chapter on Secondary Degenerations. Both of these chapters will repay exhaustive study. Intrinsic Diseases of the Spinal Cord, Extrinsic Diseases of the Spinal Cord, Meningeal Diseases and Syphilis of the Nervous System are the concluding chapters of this, the best representative of modern French neurology. When it is considered that the cost of such a work as this is only 18 francs, the reviewer is tempted to echo the hackeneyed sentiment that it should be found in the library of every neurologist of the country who has a working knowledge of the French language. JELLIFFE.

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[1] Phila. Med. Journ., March 29, 1902.

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THE
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Original Articles

SOUL PARALYSIS.*

BY H. H. HOPPE, A.M., M.D.,
OF CINCINNATI, OHIO.

LECTURER ON NERVOUS DISEASES, UNIVERSITY OF CINCINNATI, MEDICAL COLLEGE OF OHIO, NEUROLOGIST TO CINCINNATI HOSPITAL.

One of the problems in neuro-pathology and neuro-physiology is that of the higher reflex acts. The cortical reflex arc by which a spontaneous or voluntary act becomes possible, is necessarily complex. The motor section of the arc is rather simple, but the sensory is very complex. General and special sensations, skin, muscle, bone impressions, and impressions from the eye and ear especially, but even those of smell and taste, all convey stimuli to the cortical motor areas and influence motor acts. It is possible therefore to interfere with spontaneous motor acts by interfering with the conveyance of sensory stimuli or by destroying the memory pictures of sensory impressions. We know in the more simple reflex acts that the consummation of the act is interfered with if the medium which conducts the sensory stimulus is destroyed.

Does the same hold good in the sphere of the higher psychic reflex acts? Could the development of motor activity in a fully developed nervous organism be entirely prevented if it were possible to prevent any sensory stimuli from reaching the psychomotor area? Could we produce complete motor paralysis of the body by shutting off all general and special sensory stimuli? Could we by destroying, if such a thing were possible, the association tracts leading from the cortical areas for general and special sensation, to the cortical psychomotor areas, produce a complete absence of all spontaneous voluntary motor acts? We shall see that Munk answers this last question in the affirmative.

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* Read at the meeting of the American Neurological Association, Sept. 15, 16, 17, 1904.

On the other hand, is it possible to produce an apparent motor paralysis, unilateral in character, by a destruction of the avenues of common sensation which lead to the motor area, this latter and its projecting fibers being intact? Physiologists are divided on this question. Mott and Sherrington answer in the affirmative, Munk in the negative.

Charles Bell[1] in 1826 knew perfectly the regulating influence of sensation on motion and the effect of the loss of muscle sense upon voluntary acts.

In "The Human Hand and Its Functions" he says: "Without the muscle sense we should not be masters of our own bodies, we could not command our muscles when standing, much less so when walking, running or jumping, if we were not in possession of the sensation of the condition of the muscle before we projected the act of the will," etc.

As early as 1886 Munk[2] first used the term "Soul Paralysis." He produced in dogs, by extirpation of the sensory cortical area a form of paralysis very similar to what Bruns later on described as occurring in human beings.

Neurologists have put on record undoubted cases similar to the one which forms the subject of this paper, where a complete loss of voluntary and spontaneous motor power was present in cases presenting loss of sensation, central in origin, in the affected extremity.

Mrs. F. F., Aet. 47—married—3 children, one miscarriage. Has had no venereal disease. Four years ago typhoid—during third week had sudden attack of acute hemorrhagic non-purulent encephalitis with paresis of right side of arm and face, and well marked amnesic aphasia with slight paraphasia. The paresis disappeared; the aphasia, never completely. Two years later had an epileptic seizure; none since.

Four weeks ago had an apoplectic seizure without loss of consciousness. I was called to see patient four weeks after attack. In the interval she had regained some power in leg, but arm had remained absolutely helpless. She has suffered much with pain in the right side of head, neck and shoulder, occasionally in the right arm and leg. These pains occur in paroxysms and are very violent. Independent of the disturbance of speech there is at times mental confusion. Patient complains of the fact, that when she lies in bed, she does not know where her right arm and leg are. This condition worries her very much.

Examination.—Mental condition good, is cross and complaining at times, but usually happy and cheerful. Refers to her arm and leg as to a second person; constantly calls the arm or leg she or he. Is able to speak, but not fluently; has lost knowledge of nouns; recalls the names of but few of the ordinary objects about her—if given the name repeats it readily at times, at others with some difficulty, for instance, always says can't for can—forgets the name almost immediately, often shows marked paraphasia. Invariably knows the nature and use of an object. Is unable to read, but recognizes letters, spells words, but does not pronounce the word. She does not recognize the meaning of words, because letters grouped into a word have no meaning for her. She has no functional disturbance of sight, no hemianopsia. Can neither read nor write.

Sense of hearing, taste and smell are normal.

Slight paresis of lower half of right side of face and tongue.

Complete analgesia on right side of face, arm as far as elbow, and over various parts of legs and side of chest.

Loss of muscle sense and sense of position of the limb is complete. Does not know where leg and arm are when they are covered by bed clothes, in such a manner that she cannot see them.

There is complete thermanesthesia of entire side. Astereognosis is well marked.

The right arm is completely paralyzed, has not moved at all during the four weeks since onset of trouble. The arm is flaccid, relaxed, reflexes are absent, upon being requested to move the finger or hand, was not able to make the slightest movement, although she made an attempt to do so, showing that she understood the request. Very often, later on, a request to move the right hand was answered by the movement of the left hand.

If the patient, however, was told to watch the doctor's hand and then told to repeat the movement, the movement could be executed at once. Then the apparently completely paralyzed hand was made to extend and flex the fingers and thumb, to spread them wide apart, and to flex and extend the hand on the second day. Inside of a week all the normal movements of the arm and fingers —flexion, extension—moving of arm and placing hand on head and elevating shoulder could be done, usually only after she first saw the movements executed by the physician, and only then when she was told to watch the movement closely.

Patient can move the leg slightly, flex it in the knee-joint, but has made no attempt to walk. The foot is in the typical condition of foot drop. The patellar reflex is present, no ankle clonus. Babinski sign is present. The muscles are relaxed and the sensory disturbances about like those of upper extremity.

After four weeks she could make gross movements of the arm when asked to do so, extend the arm and place hand on head. But the other movement, of hands and fingers especially, were quickly forgotten and could not be executed at all, or only very slightly, unless she first saw them executed for her, then she imitated them clumsily, slowly and with hesitation. This is especially true of extension and flexion of fingers and thumb of hand and forearm and elevation of shoulder.

The leg has extension and flexion, there is typical foot drop, but this can be overcome when she sees flexion and extension of foot done, she can walk with assistance, but owing to the fact that she does not know the location of the leg, is forced to look at her foot and is constantly turning the foot in, stumbling over her own foot.' She is in constant fear of falling. With closed eyes she is unable to walk at all, although she can stand when supported. After eight months the patient is in the following condition:

Face and tongue are normal.

The arm usually hangs along side of body, semi-flexed, the hand being usually held by the other hand, only on the rarest occasions are the right hand and arm used spontaneously, the left hand being used on all occasions for the ordinary wants. By a special effort of the will patient has fair use of hand, fingers and arm. The finer movements, however, such as threading a needle, holding pencil, are very imperfect. All movements are less perfect with closed eyes or if done out of her ordinary routine. Reflexes somewhat increased, muscular power almost normal, temperature sense defective, muscle sense and sense of position of limb almost absent. Marked astereognosis.

Patient is unable to imitate with the normal arm any passive movements made in the paralyzed arm, but is able to imitate all movements of the normal arm with the paralyzed arm.

The leg is dragged in walking like a hemiplegic leg, no ankle-clonus, no Babinski; loss of temperature, muscle sense, etc. Same as in arm. Slight motor weakness. Fear of falling and hesitation in walking still present, foot drop gone.

Since Bruns (4) was the first to present this subject in a systematic way I should like to introduce a short synopsis of his case:

Patient was a male, aet. 60. Sudden onset, without loss of consciousness.

Résumé—Sensory aphasia (Wernicke), alexia, agraphia, mild paraphasia, with tendency to optic aphasia of Freund, right sided hemianopsia, right sided diminution of sense of touch, pain, muscle sense, and sense of posture. Condition of motor function: An absence of real paralysis in right arm and leg, but the right arm is not used spontaneously for voluntary acts; lies as if paralyzed along side of patient; arm is used for reflex acts, like scratching and for unconscious acts, like bracing himself when arising from a sitting or recumbent position to a standing one. When shown how to make a movement, and especially when the movement is made passively in the arm several times, the patient is able to repeat it perfectly. These movements are then apparently normal, and not clumsily made, nor is it necessary for patient to control these movements with his eye. He is conscious of this defective condition and heaps abuse upon his arm, calling it vile names, etc.

Pathological Condition.—Softening of cortex and sub-cortical white matter of first temporo-sphenoidal, parietal lobe, gyrus angularis, and posterior portion of the internal capsule. The rest of the left hemisphere, including the psycho-motor area, is intact.

There are other cases on record, which vary somewhat from that of Bruns' case.

Bleuler's case (5)—Right hemiplegia, loss of sense of location of limbs, soul-paralysis, diminution of temperature sense, right hemianopsia, amnesic color blindness, alexia, amnesic aphasia.

Pathology—Softening of left island of Reil, largest part of both central convolutions and adjacent cortex and subcortical substance.

In this case there was some motor weakness. Patient could execute movements only when seeing them.

Anton (6) has put four cases on record. In these cases the parietal cortex and the sensory tracts were affected.

Spaeth, Heyreard, von Zoemssen (7) in an article entitled "General Anesthesia" report two interesting cases. Spaeth's case fell as soon as he closed his eyes, and the other two patients ceased speaking when the ears were closed.

Spiller's case is not as typical as others above. He reports that patient had use of arm and leg, but did not use them. Autopsy showed meningitis in plaques of the parietal lobe (13).

The pathological condition underlying this peculiar phenomenon is rather uniform in all the published cases, viz.: a destruction of the cortex or the sub-cortical region of the parietal or spheno-temporal lobes, or of the sensory tracts. In Bleuler's case the ascending frontal convolution, and the island of Riel were softened, but this case presented some motor weakness.

Bruns has named this condition soul-paralysis on account of its resemblance to Lissauer's soul-blindness, which is also due to a destruction of the association tracts between the optic center and the other cortical centers. Soul-blindness can only be partial, for in order that it may occur, it is necessary that the association tracts should be destroyed in the immediate neighborhood of the optic cortical center, and this necessarily leads to more or less destruction of the center itself, and to consequent cortical blindness, with contraction of the field of vision. Theoretically, the same might occur by destruction of these association tracts wherever they occur, but in practice, such extensive lesions are not seen.

Alexia is a partial soul-blindness, the letters and words are seen, but not recognized as symbols of language.

Optic impressions, however, are registered on bilateral cortical centers, and in order to have complete soul-blindness, it is necessary to have a bilateral destruction of these association tracts, from the optic area to all of the other sensory areas. Soul-blindness occurs, therefore, when with optic tracts intact and optic centers intact, visual impressions can reach the cortical optic centers, but are not recognized, because the necessary associative impressions are not at hand, owing to a destruction of the associative tracts. Let us contemplate how objects are recognized by sight. We see a red billiard ball; we at once know that it is red, round, hard, heavy and smooth. By means of our sense of sight we see only red, and circular; in order to know that it is round and smooth, we must call into play the sense of touch; that it is hard, and heavy, the muscular sense; and that it is cold, the temperature sense; and that it is a billiard ball, our previous optic impression.

All processes of the soul are similarly complex, consisting of impressions from all sensory centers. Intended voluntary muscular movements are equally complex, their fineness, precision, and in fact, very often their origin, are dependent upon preceding sensory impressions and knowledge which are, unconsciously, utilized in each movement which is made. If, therefore, the motor center and motor tracts are intact, but the sensory centers, or association fibers connecting the sensory centers with the motor tracts are destroyed, the sensory irritations for muscular movements no longer reach the motor cortical area. There is a break in the higher sensory motor cortical reflex arc, and, a paralysis results, not due to a break in the motor section of this reflex arc, but in the sensory. Spinal reflex acts are possible. Spontaneous are not executed.

If this is true, why does not soul-paralysis occur in every case of cerebral organic hemianesthesia. In the reported cases, loss of sensation was always present, especially astereognosis, and loss of muscle sense.

As a matter of fact, in ordinary hemiplegia, with hemianalgesia and loss of thermic sense, and astereognosis, the phenomena seen in soul-paralysis are not observed. It is only when the muscle sense and the sense of posture in the limb are lost, that we observe a case of soul-paralysis. Why does this condition occur sometimes and not at others?

We all know from our clinical experiences that we meet with cases in which all the qualities of sensation are interfered with and no soul-paralysis results.

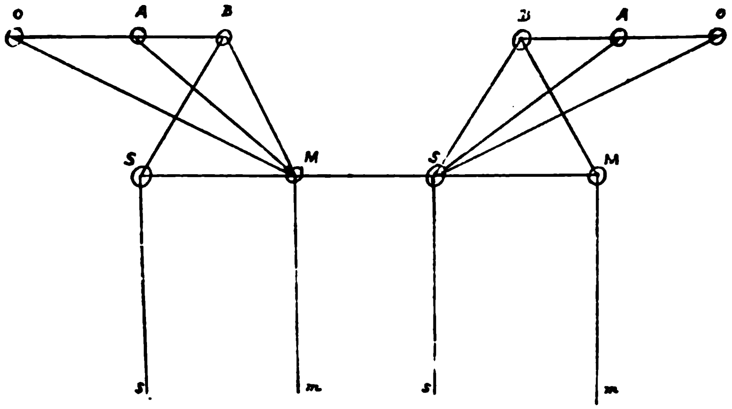
We know that loss of muscle sense alone, will not result in soul-paralysis. In Starr and McCosh's[8] case of loss of muscle sense following the extirpation of a cortical tumor, the patient presented marked ataxia but no paralysis.

Strümpell calls attention to the fact that in syringomyelia we may have a profound loss of the temperature sense, and the sense of pain, with muscle sense, posture sense, and sense of touch intact, and the patient experiences no interference with the free use of the extremity. We see thus that the loss of any of the qualities of sensation, and in the majority of instances of all of them combined, is followed by soul-paralysis.

It is necessary that there should be a destruction of the association tracts, connecting the sensory centers with the other centers, not only the motor, but the higher psychic centers. When there is a loss of all association impulses from the sensory centers, the soul is not conscious of this portion of the brain, the higher cortical reflex arc is destroyed, and an apparent muscular paralysis results.

Tracts, however, from the optic and acoustic centers and from the sensory centers of the opposite side through the corpus callosum, to the motor area of the apparently paralyzed side, are intact, and this explains the phenomena, that patients can make movements when the hand is seen, when they are commanded to make them, or when they imitate passive movements of the well arm with the paralyzed arm.

In the scheme, M m represent the motor centers and tract, and their destruction would be followed by a pure motor paralysis. Destruction of S s, the sensory centers, and tracts, would ordinarily be followed by hemianesthesia.



S s. Equal sensory tract and sensory centers.
M m Equal pyramidal tract and motor areas.
B. Represents consciousness.
O. Equal optic areas.
A. Equal acoustic areas.

B. O. A. represents respectively, the higher psychic centers, the centers for vision and hearing, if the association tracts S. B. connecting the sensory centers with the psychic centers, or according to Bruns also with the optic and acoustic centers are destroyed, soul-paralysis results.

The fact that passive movements in the well arm can be imitated perfectly with closed eyes, is due to the fact that sensory and motor impulses from the healthy side of the brain are sent by way of S. M. M. through the association tracts by way of the corpus callosum from the healthy centers of the normal hemisphere to the motor center on the diseased side. Pick calls this process hemiplegic parallelokinesie.

It seems to me that the condition resembles trans-cortical sensory aphasia, more than trans-cortical motor aphasia, although Pick's[9] nomenclature of transcortical disturbance of motion is well chosen.

The resemblance to amnesic aphasia is very apparent. In amnesic aphasia, the knowledge of words is lost, but words can be repeated. So in soul paralysis, voluntary movements are lost, but can be imitated when seen. In amnesic aphasia, the words at first are quickly forgotten, but usually a fair stock is gradually regained, so also in soul paralysis, the movements are quickly forgotten, but afterwards they occur more or less spontaneously. And just as few cases of amnesic aphasia ever regain the use of language completely, so also in soul paralysis, the movements are never completely and perfectly regained.

A condition similar to organic soul paralysis was first described, by Duchenne, under the title of "Perte de la Conscience Musculaire." Similar cases were published by Lasegue, Strümpell, Bastian, Muller and Schuman, Goldscheider and Nothnagle. The most recent exhaustive article is by Fridel Pick, in which he also discusses organic soul paralysis and calls the condition transcortical disturbance of motion.

In order to present to you the subject of soul paralysis clearly, let us briefly review the mode of origin of a voluntary motor act in the child. At the moment of birth, the child is capable of nursing and swallowing, but these are purely subcortical reflex acts.

From the very beginning, numerous impressions stream in upon the brain of the child, through all the sensory avenues. These impressions are partly registered upon the cortex, where they are stored as memory pictures, partly pass along new paths to other parts of the surface of the brain. One of these parts is the motor area which is excited by these impressions, and reacts in its specific way, namely, with a motor act. The child first pays attention, then makes a movement. In the beginning these movements are awkward, but soon become more and more perfect, definite in their intention, until finally the child reaches for and grasps an object which is held out to it. Up to a certain point this act is purely reflex, we soon notice that the child can choose to grasp or not to grasp. A psychic parallel has been added to the act. The child is conscious of the muscular act. This element of consciousness is also sensory in its origin. The child can be conscious of it through seeing the movement, and through the impressions which the movements make upon the sensory fibers of the joint surfaces, tendons, ligaments and skin. We have then the reflex act plus the element of consciousness.

In all highly developed nervous organisms the sensory impressions coming from general and special organs of sensation, need not necessarily be transformed at once into motion, but are stored away in the cortex until at the opportune moment they present themselves to our consciousness, and instigate motor acts. These latent simple impressions are worked up by consciousness into very complex thoughts. They are also capable of interacting upon each other, exciting or inhibiting and thus form the basis of our voluntary acts. When a voluntary act is performed as a result of an impulse of the will it is necessary that all the previous sensory impressions should be marshaled in their proper places, in order that the act may be performed quickly and with precision. The most important of all is that the memory picture of the act as a whole should be at the immediate disposition of the soul. Anything which interferes with this highly complex mechanism will interfere with the performance of the motor act, and we have seen that with the motor mechanism intact an apparently complete motor paralysis can result from a disturbance of the sensory section of this highly complex cortical arc. This condition has been called soul paralysis.

Recent experimental work in neuro-physiology has thrown new light on the subject of the relation of sensation to motion.

By improvement in the technique of operations, the researches in animal physiology have been better and more definite.' Mott and Sherrington's researches had somewhat startling results. They experimented on monkeys. By cutting all the posterior roots leading to the extremities of one side, they found that the movements of the hand and foot were practically abolished. The movements of the arm and forearm, of the leg and thigh were less interfered with. The arm hung useless from the shoulder, adducted, flexed at the elbow, and at the wrist—just as an arm that is carried in a sling; the leg was carried adducted and flexed, so as to free the foot from the floor when the animal ran; in climbing, both arm and leg were allowed to hang helplessly, and were not used. When allowed to become very hungry, the good arm being secured on the back, no attempt was made to take the food with the right hand, but the neck was craned forward, and the food grasped with the mouth. If the food was put in the anesthetic hand, no attempt was made to bring it to the mouth; the animal seemed unable to reach the hand to the mouth, even when urged to do so.

Interesting in the Mott-Sherrington monkeys is the distribution of the motor disturbances. It increased in proportion to the distance from the attachment of the affected extremity, being less marked at the shoulder and hip-joints, more marked at the knee and elbow, and motility was practically abolished in the hand and foot, especially so in fingers and toes. In this respect the disturbance of motion resembled that which follows the removal of the psycho-motor area, except that it was even more pronounced. When making exceedingly violent resistance movements the monkey occasionally used the fingers and toes, but never more than once or twice. As a result of these experiments Mott and Sherrington have come to the conclusion, that after loss of sensation in the extremities, the associated movements are not very much affected, but that those muscle-groups which are used for making the finer movements, complex, individual, and voluntary movements, movements for definite purposes (just those groups which are mostly represented in the gray matter of the psycho-motor area), are grossly interfered with, and practically abolished. They have come to the conclusion that the will is no longer able to make use of the muscle groups. After sensation has been abolished, the animal can no longer use the hand for grasping, and bringing food to its mouth, or use the foot and leg for climbing. Mott and Sherrington consider the lesion a permanent one, and conclude that not only the sensory centers in the cortex, but the entire sensory tract to the periphery takes part in the voluntary acts of the body, and its integrity is necessary for the accomplishment of these voluntary acts; that centripetal sensory impulses, especially from the palm of the hand and the sole of the foot are necessary for the execution of the highest complex movements. They call attention to the fact that the animal *could* not perform these acts and not that it did not want to. They hold that this was the result of the fact that the animal was no longer able to form an idea of the act which it should perform.

Interesting in connection with this paper and a corollary to Mott and Sherrington's experiments on animals is Strümpell's[11] published case of total anesthesia of the right forearm and hand following a stab wound of the spinal cord. The defects are almost identical, at least as far as the hand and forearm are concerned, with published cases due to cerebral lesions. As a result of his case Strümpell concludes that the total loss of sensation renders the following voluntary acts impossible.

I. The limitation of a movement in space to a given degree (for instance, bending the forearm to a given angle).

II. Continued static fixation of the limb in a designated position (viz.: static coördination).

III. Rythmic, equal, slow movements to an extremity.

IV. Limitation of voluntary movements to isolated muscles, for isolated movements, viz.: movements of one finger with quiet fixation of the others.

V. The execution of consecutive movements, one after the other in proper order.

VI. The execution of complicated movements which call into play various muscle groups, which must act together, viz.: intended movements, occupation movements, writing, etc.

Strümpell holds that all these defects are atactic in character.

All these defects of movements are improved by optic coördination, but the latter can never replace the muscle-sense, etc., completely, for even with open eyes the above movements are defective.

Munk[12] repeated the experiments of Mott and Sherrington, and found immediately after the operation the same results as the English investigators, but with this difference—Mott and Sherrington report, that after four months, the practical abolition of motion of hand and foot, remained unchanged, whereas Munk found that even on the very day of the operation, in some monkeys, but practically in all after a few days, that after persistent irritation, the animal soon began to use the anesthetic hand for voluntary isolated movements; that this use gradually became more and more constant; that although the animal always preferred to use the uninjured arm, it quickly and spontaneously used the anesthetic hand and arm when urged to do so; that it used this arm and hand for purposes of eating, and of cleansing itself, usually using it to assist the good arm and hand rather than using it in isolation, but that it would use it singly, and with a motive; but this improvement progressed only to a certain degree, and that the movements of the anesthetic arm were always characterized by an exaggeration, lack of precision and an awkwardness, which contrasted strongly to the well arm. He found, however, that the voluntary associated movements, such as are necessary for climbing, walking, running, were permanently lost or if any improvement were gained, it was of such a character and degree as to be of very little use to the animal.

He explains the difference in his results, in so far as they concern the voluntary isolated movements of the anesthetic arm, in his experiments as to those obtained by Mott and Sherrington, by the fact that the animal can make the movement, but does not wish to make it. If the food is so placed that the animal misses it in an attempt to reach it, it concludes that the movement is useless, and no longer attempts to make it. But if the food is placed in the anesthetic hand, and the monkey, as a result of the combined use of hand, and a stretching of neck and head forward, succeeds in placing the food in its mouth, it finds that the movement is successful, and continues to use the hand to bring the food to its mouth, and gradually becomes more and more proficient in the use of the anesthetic hand and arm.

Munk has thus shown quite a difference in the result of his experiments from those of Mott and Sherrington. That after resection of the posterior roots, voluntary associated movements, such as are seen in walking running and climbing, are permanently lost, whereas, voluntary isolated, individual movement, made with special intent, are gradually regained, but never to a state of perfection or precision as before the operation. He has shown that just the opposite condition is found after extirpation of the "Fuhlssphere"—viz.: that isolated voluntary intended movements are permanently lost, whereas voluntary associated movements as in walking, climbing, running are retained, although with some awkwardness and lack of precision.

Munk claims that the conclusions drawn by Mott and Sherrington are not justified by the results of their experiments, that not only the cortex, but entire sensory tract from periphery to cortex, is active during the execution of voluntary isolated movements, he denies the law, that the loss of sensation leads to the loss of voluntary muscular acts; that while this law is true for the entire animal, and all its acts, it is not true for isolated extremities.

Munk agrees, however, that the integrity of the sensory tract, and the cortex are necessary for precision, and perfect adaptation of a muscular movement to the end intended, but not for the execution of the act itself, the voluntary act can be performed, but it lacks fineness and precision, even though there be complete anesthesia.

Munk explains the loss of isolated acts after cortical extirpation, on the ground that small definite areas of the cortex are necessary for their performance, but that associated movements, like walking, climbing, etc., can be performed because larger areas are called into play; they are done awkwardly and clumsily, because the cortical terminations of the sensory tract, and the beginning of the motor tract being destroyed, the centers are deprived of the exact knowledge of position of muscles and joints, as well as the nature of the floor or tree to be climbed, or the food to be grasped. He sums up the result of the loss of sensation on motion as follows:

The results are twofold, and are based upon two grounds.

All movements of the anesthetic extremity, which are the immediate result of a reaction to irritation of the sensory stimulations are no longer carried to, nor are perceived by the central nervous system. Permanently lost, therefore, are all proper reflex movements, the common reflex movements, and the cortical reflex movements of the affected extremity.

All other movements of the extremity are damaged, are performed awkwardly, and clumsily, because of the lack of proper irritation of the cortex, by the sensory tract. Cortical, as well as spinal centers, are kept in a state of constant irritation, constant tension, constant tone, by communications brought to them from the extremities, even when in a state of rest, by the sensory fibers from skin, muscle, ligament and bone. The cortical motor centers are kept in a constant knowledge of the exact state of contraction of muscles, the position of limb, condition of joints, etc. This knowledge is necessary for prompt, accurate, and perfect executions of isolated as well as associated muscular acts, and when it is wanting, the act becomes awkward, imperfect, and incomplete. This is especially true of well ordered coördinated finer movements.

If this knowledge is wanting entirely, as it must be when there is total loss of sensation, the limb is not used, because the cortex of the brain has temporarily no knowledge of its existence, and hence, no attempt is made to use it. We know, however, that the sensory paths are reëstablished when once destroyed, and other areas of the cortex take on sensory functions, and therefore some use of the limbs returns.

My clinical case corresponds with Munk's physiological observation. The finer movements were regained, it must be admitted never perfectly, while the associate movements, except for walking, have not made much progress.

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ANATOMICAL FINDINGS IN TWO CASES OF KORSAKOFF'S SYMPTOM-COMPLEX.

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In 1887 Korsakoff published his first descriptions of mental disturbance associated with multiple neuritis. Since then much has been written about this symptom-complex; but comparatively few cases with anatomical findings have been reported.

The following cases, which occurred in the medical service of the Boston City Hospital, are apparently of alcoholic origin and died in an early stage of the disease.

The first case is an example of an acute polyneuritis with a chronic mild delirium, developing symptoms resembling cerebral hemorrhage. The second case is more chronic in on set and more typical of Korsakoff's symptom-complex. Both exhibit marked changes in many parts of the nervous system.

I wish here to express my obligations to Dr. J. W. Bartol and H. D. Arnold for the use of their clinical records. The autopsies were performed at the Boston City Hospital during the pathological service of Dr. F. B. Mallory. I wish also to thank Dr. E. E. Southard and Dr. A. M. Barrett for suggestions as to the anatomical interpretation.

CASE I. (FROM SERVICE OF DR. J. W. BARTOL.)

S. S., a woman, age forty-eight, married, cook, was admitted to the Boston City Hospital, September 10, 1903. Family history was negative. Patient had no children. There were two miscarriages, no cause known. She had slow fever and measles as a child, also asthma. For several years she had been addicted to liquor, and for the last few months had taken two or three glasses of whiskey daily. She considered herself well up to two weeks previous to admission. At that time she commenced to have pains in her knees and ankles, and later she became unable to walk. Her legs were often swollen at night. She was a well-developed and obese person. The pupils reacted promptly to light and in accommodation. Tongue was clean. There was a systolic murmur of the heart, the pulmonic second being accentuated. Lungs were negative. In the extremities there was no swelling or tenderness of the joints, but some tenderness of the muscles. Knee-jerks were absent.

Sept. 14.—Some delirium at night. She was talking considerably, frequently attempting to get out of bed. There was marked wrist-drop, with inability to use the fingers.

Sept. 22.—Temperature remained normal. Patient was sleeping fairly well. Frequently woke up and called for various friends, thinking that she saw them. Complete toe-drop and wrist-drop.

Sept. 24.—There was noted a convulsion with twitching of the face, neck, and shoulders of both sides; more prominent on the left. This convulsion lasted about 45 minutes. She was unconscious and could not be aroused. After this the mental condition was the same as prior to the attack. She remained quiet the rest of the day and night. On the following day there was another convulsion, in which both sides of the face, abdominal muscles, and the legs to a slight extent, were affected.

There was spasticity of the right arm. She was unconscious with incontinence. This attack lasted about four hours; was quieted with morphine.

Sept. 26—She was in a stuporous condition from which she could be fairly easily aroused. She recognized her name and contradicted the physician on being called by her wrong name. During the afternoon and night, when disturbed there were noted explosions or clonic spasms of the face, and to a less degree of the body. The labio-nasal fold was indistinct and there was a decided droop to the left side of the face. Tongue was protruded to the left. There was flaccidity of the left arm and leg. She seemed to recognize her husband. She kept up considerable talking prior to the convulsion. Pupils were small, irregular in outline, and reacted to light. There was deviation to the right of both eyes. Pulse and temperature commenced to rise.

Sept. 30.—Mental condition was much the same. There were times when she was more sane and made clever replies to questions. She showed a tendency to fabricate, frequently speaking of incidents which probably had occurred in her past life. Respirations were normal. Pulse was 160. Temperature had risen to 104.5°. She began to vomit and was unable to retain any nourishment. Mental condition gradually grew worse. She rapidly became more feeble, and died Oct. 1, 1903.

Clinical Summary.—Acute alcoholic multiple neuritis in a woman of forty-eight. Accompanied by delirium, hallucinosis, and romancing. Later convulsions with twitchings of various muscle groups, which was followed by spasticity of one extremity and flaccidity of the extremities of the opposite side. Some paralysis of the facial muscles. Death after five weeks, preceded by a rapid rise in pulse and temperature.

Anatomical Findings.—Autopsy five hours post-mortem (Drs. R. E. Lee and E. E. Southard).

Well developed, obese white woman.

Peritoneal Cavity—Mesenteric lymph nodes normal.

Pleural Cavity—Normal.

Heart—Weight, 330 gms. Subepicardial fat increased in amount. Myocardium normal in color and firm. Endocardium normal. Mitral valve slightly thickened at the edges. Slight amount of sclerosis seen back of the cusps of the aortic valve.

Measurements—Tricuspid valve 11 cm.; pulmonary valve 7 cm.; mitral valve 11 cm.; aortic valve 7 cm.; left ventricle 1.75 cm.; right ventricle 3.24 cm.

Fresh specimen examined with acetic acid shows no fat.

Lungs—Left dark red and edematous. Posterior part of the upper lobe is firm and contains little air. Cut surfaces granular. Small greenish bronchial plugs of pus exude on slight pressure.

Right Lung—Dark red and moist. Posterior part shows the same process as in the left lung, but not so marked.

Spleen—Weight, 55 gms. Capsule ruptures easily. Trabeculæ not increased. Malpighian bodies seen with difficulty.

Gastrointestinal Tract—Normal.

Liver—Weight, 1760 gms. Rather yellow in color, greasy to the feel. Central veins injected. Surface of the liver slightly roughened and adherent at several points to diaphragm and adjacent viscera.

Kidneys—Weight, 210 gms. Normal. Fresh specimen examined with acetic acid shows no fat.

Brain—Weight, 1,100 gms.

Head—Scalp and calvarium normal. Dura everywhere free and tense. Brain appears to retract slightly on removal of skull-cap. Arachnoidal villi moderately developed. Pia almost dry. Vessels normal. Veins of sulci moderately injected. Substance normal. Ventricles contain a normal amount of fluid. Inter-brain, mid-brain, and pontine region normal on section. The medulla cuts well, except in ill-defined fusiform area of slightly reduced consistence and slightly pinker tinge to the left of raphe in the formatio reticularis a few mm. dorsal to the olive. The left side of the medulla is slightly larger than the right and the olive seems pushed ventrally by 1-2 mm. Remainder of bulb and cord normal.

Microscopical Findings.—Lungs—Marked congestion in places with more or less hemorrhage. Some foci of exudation containing polynuclear leucocytes, serum, fibrin, and red blood corpuscles. Pigmented mononuclears are fairly abundant in places. A variety of organisms are present.

Spleen—Not notable except for much brownish pigment in coarse granules within phagocytic cells.

Liver—Shows marked fatty infiltration which is least abundant adjoining the portal vessels. There is some increase of portal connective tissue with formation of so-called bile ducts.

Kidney—Shows atrophyof numerous glomeruli and tubules and increase of connective tissue. Moderate congestion of the vessels in the pyramids.

Thyroid—Many of the glands are dilated and filled with colloid material. There is some increase of connective tissue. Many foci of lymphoid and plasma cells.

Peripheral Nerves—With Marchi method intense degeneration of internal and external popliteal, the ulnar and musculospiral nerves of both sides. Both anterior crural nerves and median nerves (at the elbow) show many degenerated fibers, but not so marked as the above. The brachial plexuses have a few affected fibers, more marked on the right side.

The vagi and phrenic nerves present numerous degenerated fibers. Other cranial nerves were not examined.

Cord—Anterior horn cells of the lumbar, sacral and cervical enlargements show typical acute changes of the axonal type. There is considerable excess of yellowish granular pigment arranged peripherally, usually occupying the basal portion.

The lumbar and sacral segments are most severely involved. Most of the cells of the lateral group are affected. The mesial are usually well preserved, showing abundance of large staining masses (Nissl).

In the dorsal segment a few motor cells show marked excess of pigment.

Most of the cells of Clarke's columns present axonal reaction of a severe grade.

In the medulla and pons there are marked changes of similar type, in the cells of almost all the cranial nuclei, except the hypoglossal. In the vagus nuclei from four to six cells in each section show stages of central chromatolysis.

Many cells in the descending root of the trigeminus, also the cochlear, and glosso-pharyngeal muscles, are affected.

Marked axonal reaction involving most of the cells in the facial nuclei. The cells of the inferior olives, and of the nuclei of Goll and Burdach present marked changes.

Cortex—The pia is quite generally thickened and shows a moderate cellular infiltration. Blood vessels of the cortex are considerably injected. In places a slight perivascular infiltration, chiefly of lymphoid cells. No marked changes of the walls.

Various areas of the cerebral cortex after fixation in alcohol were prepared by a modified Nissl method (paraffin sections). Throughout the cortex there is a general mild degree of so-called acute alteration of the cells. The pyramidal cells are swollen. The processes are visible farther than usual from the body of the cell.

The stainable substance is disintegrated into a pale, fine granular substance. In a few cells only, can one see the Nissl bodies at all and in these instances they have a fragmented appearance.

The nucleus is greatly swollen and the nuclear network quite distinct. The nucleolus often stains in a metachromatic way. In many nuclei there are coarse granules staining deeper than the nuclear network.

The Nissl bodies of the Betz cells are fairly well preserved. There are no marked glia changes.

With Marchi method there are found no notable degenerations in the cortex.

In the medulla considerable degeneration is seen in the restiform bodies, also a few fibers in the posterior longitudinal bundle, and the fillet are blackened.

Throughout the cord there is some marginal speckling, most marked in the direct cerebellar tract, and there are a number of blackened fibers in the middle of the posterior columns. Numerous fibers are affected in both the anterior and the posterior root bundles.

Summary of Anatomical Findings.—Slight arteriosclerosis. Hypostatic pneumonia. Fatty infiltration of the liver. Acute degenerations of many of the peripheral nerves. Axonal reaction in cells of the anterior horns; Clarke's columns and many cranial nerve nuclei. Degenerations in the posterior columns, direct cerebellar tracts, and the root bundles. Moderate "acute alteration" of the cortical cells.

CASE II. (FROM SERVICE OF DR. H. D. ARNOLD.)

H. B., travelling salesman, age about thirty-five. Family history negative. History of several slight attacks of rheumatism, and gonorrhea twice. Patient, at least for the last few years, has been an excessive drinker. Up to about a year ago he drank beer chiefly. He was persuaded to abstain entirely for about six months on account of chronic urethritis. Then he commenced to drink harder than before, taking several glasses of whiskey daily. Frequently was incapacitated for work. He began to have trouble with his feet in May, 1903. He complained of pain in the great toe of the right foot. This gradually extended to the other foot. His feet became swollen and he often cried with pain. Soon after the onset of this trouble his friends noticed a marked change in his disposition. He became very irritable and whimsical, and felt a personal affront when his opinion was crossed. He would often cry because of the supposed injury to his feelings. He became restless, quite secretive, and even suspicious. Formerly he had been quite open and communicative. It was noticed also that his memory was becoming poor. Many things of recent happening he was unable to recall. At one time he was consulted and agreed to a change of policy on the part of the company which he represented. A few weeks later when this change went into effect he was very bitter because he had not been consulted, denying all knowledge of the event. During the last six months he had frequently contracted bills and borrowed money, but later proved unable to recall the occurrence. This memory weakness increased until at the time of admission to the hospital he was unable to give a very satisfactory account of the last months of his life. His feet began to bother him so much in September that he finally consented to go to the hospital. He was admitted to the Boston City Hospital Sept. 29 1903.

His general condition was good. Muscular tremors were seen in hands and tongue. The pupils were normal. Heart and lungs were negative. The plantar surfaces of both feet were somewhat swollen, reddened and tender, particularly over the metatarso-phalangeal joints, which were extremely painful. The knee-jerks were lively. There was no atrophy or edema.

Oct. 3.—The condition of the feet had improved. The patient was fairly quiet, less tremulous, and sleeping about half the night. Temperature was normal.

Oct. 7.—Up and out of doors daily in a chair. Slight tremors. He slept six hours. There was less pain and swelling in the feet. Numbness in the right great toe.

Oct. 11.—Pain was more intense and tenderness more marked. No pains in the legs. General condition remained good.

Oct. 19.—Foot symptoms were the same. He complained of pains in the calves of his legs and was nervous at night. He was somewhat irrational and less thoroughly aroused. For several nights previous he had been getting out of bed and walking about the corridor, apparently not realizing, for the time being, his whereabouts. He slept about half the night.

Oct. 24.—He was irrational and delirious, required constant watching. Condition of feet seemed improved. He slept little. A day or two later became semi-comatose and unable to articulate and to swallow. There were no convulsions, no spasticity. Pupils reacted promptly to light. No paralysis, but incontinence of sphincters. Heart was rapid, irregular; sounds somewhat distant. No murmurs. Lungs showed occasional moist râles in the back. Knee-jerks were equal and normal. There was no Babinski reflex. He gradually became more and more somnolent, with increased difficulty in breathing. Death occurred Oct. 30, apparently due to failure of respiration.

Clinical Summary.—Acute alcoholic mental confusion following chronic neuritis, and of the type of Korsakoff's symptom-complex. Marked amnesia for recent events, irritability, increased emotional reaction, imperfect orientation and mild delirium.

Later developed difficulty in speaking and in swallowing. Death from vagus paralysis and failure of respiration.

Anatomical Findings.—(Autopsy by Drs. C. W. Keene and E. E. Southard).

Fairly developed, well-nourished white male of excellent musculature.

Peritoneal Cavity—Vessels of intestines are injected. No fluid. Many bubbles of gas show in mesentery of sigmoid flexure and descending colon. Culture shows bacillus aerogenes capsulatus.

Pericardial Cavity—Contains about 25 cc. straw-colored, serous fluid.

Pleural Cavities—Few fibrous adhesions lower left lobe to back and diaphragm.

Heart—Weight, 390 gms. Subepicardial fat greatly increased, envelops whole organ. On section, dark red color, rather firm, slightly granular in appearance. Fresh sections stained with Scharlach R., show very slight fatty change.

Few small, fairly firm, elevated, white patches in coronary arteries. One in anterior coronary covers vessel wall for 2.5 cm. from mouth.

Measurements—Tricuspid valve 12 cm.; pulmonary valve 7.5 cm.; mitral valve 10 cm.; aortic valve 7.5 cm.; left ventricle 2 cm.; right ventricle 0.6 cm.

Lungs—Fill cavities. Lower lobes dark red; upper shade to grayish pink showing black carbon mottling. Both crepitant throughout, but darker portions are slightly firmer to touch. On section dark parts drip blood; slight amount of muco-pus shows in bronchi.

Spleen—Weight, 145 gms. Very dark red, fairly firm, with rather loose capsule. On section, trabeculæ and Malpighian corpuscles clearly made out.

Liver—Weight, 2320 gms. Reddish brown color, on section red mottled and small drops.

Kidneys—Weight, 356 gms. On section dark red. Capsule strips easily leaving smooth surface. Cortex 6-7 mm. Pyramids distinctly marked. High blood content. Section stained with Scharlach R. shows slight fat.

Gastrointestinal Tract—Normal but for injected blood vessels.

Genital Organs—Right testicle threads out well. Left does not thread. Tubules cannot be made out and glandular tissue is replaced by opaque grayish, fibrous material, rather firm to touch, except at upper part of testicle.

Aorta—Shows many small, 2mm., elevated, fairly firm, yellowish white patches in ascending portion; several small ones around mouths of arteries in descending portion.

Head—Hair fairly thick and black. Scalp veins distended with fluid blood. Calvarium heavy, without diploe. Several deep excavations reaching to outer table, to receive groups of arachnoidal villi without dural sheaths.

Dura—Lax, nowhere adherent. Middle meningeal vein system injected. Sinuses filled with fluid blood, Arachnoidal villi slightly developed except at a few foci along longitudinal sinus.

Pia—Contains a moderate amount of clear fluid, especially at the vertex. The superficial cerebral venules are injected. Veins of sulci moderately injected. Vessels at the base surrounded by edematous fibrous tissue of normal depth. Circle of Willis normal.

Brain—Weight, 1510 gms. Substance pinkish gray. Consistence slightly softer over left vertex than elsewhere: the gyri here are somewhat wider than the gyri on the right, and are only roughly symmetrical with these.

Microscopical Findings.—Heart and lung—Not remarkable.

Spleen—Much congestion. Slight increase in reticulum. A few phagocytic endothelial cells. Occasionally vacuolated. Many eosinophiles and few polynuclear leucocytes. Much pigment. Arteries slightly thickened.

Liver—General vacuolation, which is most marked at the lobule centers. Foci of lymphoid and plasma cells with slight increase of periportal connective tissue. A few eosinophiles seen.

Kidney—Tubules in general are slightly dilated, contain granular material, and show flattened epithelium. The glomerular tufts are injected. Some tubules stain more intensely with eosin than others (hyaline). Very few vacuoles. The pyramids are injected and a few lymphoid and plasma cells are seen. Arteries very slightly thickened.

Testicle—Section shows some areas in which connective tissue increase has replaced tubules, but more generally there are areas of increased connective tissues containing atrophic tubules. Spermatogenesis sluggish in better preserved areas. Foci of cells of lymphoid type are seen. Arteries are thickened.

Nervous System.—Peripheral Nerves—The posterior tibial and external popliteal examined by Marchi method show numerous degenerated fibers in nerves of the right side. The nerves of the left side not so sevérely affected. Both vagi show the Marchi reaction of nearly one-half the fibers. No marked changes in optic nerves. Vessels show some intimal thickening. No cellular infiltration.

Cord—The anterior horn cells of the lumbo-sacral enlargement have acute changes, about one-third of the cells present early stages of the axonal reaction. There is slight swelling of the cell body and atrophy of dendrites with a varying degree of central chromatolysis and eccentric nucleus. One side seems to be more severely affected than the other. An occasional motor cell in the dorsal cord and one or two cells in sections from the cervical enlargement show a similar reaction.

In the cells of Clarke's column one may find two to four cells in a section, showing dislocated nuclei.

Nissl preparations from several layers of the medulla and pons present changes of the same type in cells of some of the cranial nerve nuclei. A large number of cells of the glossopharyngeal nuclei and the motor nuclei of the vagi show severe axonal reaction as well as many cells in the nuclei of the posterior columns.

Cerebral Cortex—Fibers of the pia are considerably separated by edema. In places a slight fibrous thickening and slight cellular infiltration. The vessels are all dilated and engorged.

Pieces from various parts of the brain after fixation in formalin were stained for general histological work, neuroglia and fat. The most marked changes were found in the frontal and paracentral convolutions.

In the paracentral convolutions there is a normal distribution of cells. The first glance at the specimen shows axonal reaction of the Betz cells. In about one-half there is a fine powdering of part of the stainable substance, affecting chiefly the center of the cell, but it may extend to the periphery especially the basal part. None of the cells is completely involved. The nucleus is frequently found displaced to the side or the upper extremity of the cell. There is a little pigment deposit in many of the cells. Many of the larger and medium-sized pyramids present similar alteration.

Occasionally there is found a large pyramid which is evidently necrotic. By the Nissl method the staining is very faint, the whole cell is much shrunken, and has irregular indistinct outline. The nuclear outline is not made out, but the nucleolus remains deeply stained.

In the layer of larger pyramidal cells there is a moderate increase in free nuclei in the ground substance. Many of the larger cells are beset with satellite cells. Frequently as many as six or seven may be counted around one cell, perhaps indenting it in one or two places.

A few of the glia cells in the neighborhood of the affected Betz cells are caught in mitosis. They are almost entirely of the earlier skein forms. One or two of aster type noted.

In the frontal cortex there are a few cells which show axonal degeneration. A few of the larger pyramids have a rarefied apex, and the nucleus is pushed to one side. Satellites are not so abundant.

There are no notable changes in the cerebellum.

Blood Vessels—There is a very marked general vascular engorgement of the brain and cord. A number of small microscopic hemorrhages are found, confined to the cortical gray matter of the frontal, parietal and temporal lobes. Occasionally two or three phagocytes engorged with pigment, together with free pigment, are seen.

The vessel walls do not present marked changes. In some the nuclei of the intima seem more prominent than usual, and there is an occasional area in the cortex with slight infiltration of lymphoid cells.

Sections prepared by the Marchi method show numerous degenerated fibers in the white matter of the motor cortex. Also many fibers in the frontal and temporal areas, but not so marked as in the paracentral region. The occipital lobes are comparatively free.

In the walls and around most of the larger vessels there is a large amount of degenerative product staining with osmic acid. In the medulla there is a slight speckling of the pyramidal tracts, also the fillet and posterior longitudinal bundles. More marked speckling is seen in the cerebellar afferent tracts.

Throughout the cord there is slight marginal speckling, more marked in the direct cerebellar tracts. The pyramidal tracts also show numerous blackened fibers. There is very slight degeneration in the posterior columns, except in the lumbo-sacral region, where there is quite intense degeneration, confined chiefly to the middle of one side. A few fibers in the anterior root bundles and numerous fibers in the posterior roots are affected.

Summary of Anatomical Findings.—General arteriosclerosis involving the aorta and coronaries. Fatty degeneration of the heart, liver, and kidneys. Acute bronchitis. Acute degenerations in the peripheral nerves of the lower extremities, and also in the vagi. Axonal reaction in cells of the anterior cornua, in Clarke's columns, some cranial nuclei, and the Betz cells of the cortex.

Vascular changes in the cord and cortex, with numerous microscopical hemorrhages throughout the cerebrum. Acute degeneration of the cortical radiations, and of both motor and sensory systems of the cord, as well as degenerations of the cord not easily reconcilable with the systemic changes.

In looking over the literature of Korsakoff's symptom-complex we find as a rule distinctly demonstrable changes in the brain of cases which have been studied anatomically. These changes are variable and not specific. In an analysis of these studies it is important to keep in mind the stage of the disease.

In an early stage changes are similar to those found in delirium. In most of the more chronic cases extensive degenerations (Marchi) have been found in the white substance of the cerebrum. Several authors found these degenerations more intense in the frontal, and especially in the central, convolutions.

In cases dying early in the disease these changes are often missed. Heilbronner reported two cases of fourteen days and six weeks respectively in which no demonstrable degenerations were found. The relation of the blood vessels to this condition is important. Marked congestion and prominence of the vessels have been very commonly seen. Few have found changes in the walls, Gudden has one case in which he found glass-like thickening of the walls.

Boedeker and Cole have reported cases with thickening of the vessel walls. A tendency to minute hemorrhage and blood extravasations in varying numbers has been frequently seen. These blood extravasations occur chiefly in the gray matter of the basal ganglia and around the third ventricle. Several authors mention the resemblance of some cases to polio-encephalitis.

Softening in the cerebellum was seen in one of Wernicke's cases. Gudden in two cases demonstrated foci of softening in the walls of the third ventricle, the basal ganglia and also the pons. Cramer and Wright also report cases.

Cortical cell changes are variable. "Axonal" reaction of Betz cells is described in cases of Faure, Wright, and Cole. In some of Bonhoeffer's cases there were changes in many of the cells, showing characteristics approaching those of Nissl's "acute alteration." In other cases there was evidently typical axonal reaction. Glia changes are found in most severe cases. Nearly all the cases of Korsakoff's psychosis reported show spinal cord changes. These are especially marked in the posterior columns and in the direct cerebellar tract, where there are ascending degenerations. In more severe cases degenerative changes are often found in the pyramidal tracts, and also degenerative changes, often more observed in the anterior and posterior roots. Heilbronner found that in the cord the affected areas agreed with the clinical and anatomical neuritis.

The two cases here studied show, upon an alcoholic and arteriosclerotic basis, acute degenerations of many peripheral nerves, axonal reaction in anterior horn cells, in cells of Clarke's column, and in some cranial nerve nuclei, and various non-systemic degenerations of intraspinal fibers, together with acute or axonal alterations of various cells of the cortex. In one case proliferating glia cells are found in the neighborhood of altered Betz cells of the motor cortex.

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A CASE OF MYASTHENIA GRAVIS WITH AUTOPSY.*

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(From the William Pepper Clinical Laboratory,
Phœbe A. Hearst Foundation.)

This case is of pathologic interest because it adds another to the relatively large, though absolutely small, number in which the thymus gland was either persistent in the adult, or persistent and diseased, and associated with lymphoid infiltration of the muscles. It is interesting clinically because the patient exhibited visual symptoms most frequently mèt with in, and at one time regarded as pathognomonic of, hysteria; namely, contraction of the fields of vision, partial reversal of the red and blue fields, and later color confusion. Similar disturbances of vision have not been reported in any other case. In not a few, however, the fields were not taken, the writers simply stating that vision was normal. In the first case which I reported the fields were concentrically somewhat diminished, but there was not reversal. Myasthenia gravis is so almost exclusively a motor disease that I am not at all sure that the visual symptoms in this case were more than coincidental. The patient presented no other hysterical symptoms, the course was typical of myasthenia, and the result was death. That reversal of the fields may occur in organic disease and in functional troubles other than hysteria is of course well known.

The history of the case is as follows. The patient was a white unmarried brakeman, thirty years old. His father had died from a gunshot wound and his mother in childbed. He had had malarial fever in 1894 and gonorrhea about ten years ago, but never syphilis. He had always been robust. He first visited Dr. Stahl, through whose courtesy I saw him, and by whose kindness I am permitted to make this report, on October 14, 1900, complaining that for about two months he had

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* Read at the Philadelphia Neurological Society, Feb. 23, 1904.

had a great deal of headache, that about October the first the right eye began to droop, especially in the evening, and that at the same time the muscles of the jaw grew weak, the weakness soon spreading to the shoulders and arms, so that he had trouble in putting on his coat, shaving and buttoning his collar. At the same time vision became slightly blurred and there was some napeache and quite a little vertigo.

On examination he looked like a muscular, well-nourished man. He walked well with good firm tread, but could not raise the arms above the shoulders and any movement of the arms or hands caused great tire. Sensibility to touch, pain, and temperature was normal. The knee-jerk was present and slightly more marked on the left, but not spastic on either side. There was neither ankle clonus nor Babinski's jerk. The irides reacted promptly to light and with accommodation. He complained of diplopia. The bladder and rectum were under complete control. Speech was slow, the voice weak, and it tired him greatly to talk, but there was no aphasia or vocal palsy. When sitting upright he could swallow well, but if he leaned forward solid food stuck in his throat. He could swallow liquids without difficulty. His mental state was good. The lungs were normal. There was a blurring of the first mitral sound and the second was sharp and somewhat accentuated. The aortic sounds were normal. The area of cardiac dulness was not enlarged. He was sent to Dr. Stahl's wards at St. Agnes Hospital and for a time his muscular weakness varied greatly from day to day and even at different times of the same day, but the general trend was constantly downward. For example, by November he had great trouble in swallowing, and liquids were regurgitated through the nose. By November 20 he could scarcely get out of bed and could walk only a few halting, staggering steps. All movements of the arms were very weak and after a few efforts they would fall helpless by his side. He could hold his head up for a few moments only. In fact all effort so tired him as to cause a total though transient inability to contract the muscles he had just used. I am not sure, but think, that the use of any one group of muscles produced some weakness in others. There was no ataxia. The knee-jerk was very capricious. Sometimes a blow upon the tendon would cause a slight jerk, sometimes not. Several taps might cause no movement and then a final stroke produce a slight response. The knee-jerks were never exaggerated. At rest there was marked ptosis, most marked in the right eye. He could open the eyes widely but could not hold them so. There was but little, and soon no, power of voluntary contraction of the frontal muscles. He could move the tongue in all directions. The face was quite expressionless at rest, smoothed out, masklike. I did not notice the nasal smile mentioned by Gowers. There was no muscular wasting in the face, tongue, or extremities. There was some tremor of the tongue on extension. He could turn the head, lying in bed, much better to the left than to the right. Faradic response was slower than in a healthy person. Sensibility was still preserved and he still controlled the bladder and rectum well. For some days before death he could swallow only a little mushy and liquid food. There was no fever at any time. On November 23, 1900, he began to complain of difficulty in breathing, or rather of a sense of suffocation. This continued till November 27, when, still conscious and mentally clear, he died.

His eyes were examined several times by Drs. F. M. Perkins and P. A. Bly. The fundi were normal. At the first examination on October 29 there was some contraction of the right field with, in places, an extension of the red field beyond the blue. The left field was at this time practically normal. On November 10 both fields were quite a great deal contracted, but the red extended beyond the blue in only one place in the right field. On November 17 and thereafter he confused colors a great deal, calling red blue and sometimes white, and blue white. This was not due to speech defect or mental hebetude, but to inability to appreciate color. There was some weakness of some of the extra-ocular muscles, but the notes of this part of the examination were lost and no positive statement can be made as to the muscles affected.

The urine was normal through the course of the disease.

The necropsy was made the same day the patient died by Dr. Joseph Walsh, the pathologist to St. Agnes Hospital. External examination of the body revealed nothing, and on cutting through the skin there was found the normal amount of fat. The spleen was of normal size and consistency but on section the lympth follicles seemed a little more striking than usual. Microscopic examination showed no disease. The kidneys were a little enlarged and congested, and microscopic sections showed hyperemia and slight cloudy swelling. The adrenal glands were normal. On opening the thorax the thymus gland stood out prominently. It weighed 22 G. It contained an encapsulated chronic abscess. (Dr. Walsh informs me that this was not the creamy fluid found in degenerating thymus glands, but a true abscess.) The pus was cultured on agar and bouillon and smears were made. The smears showed no organisms with the common stain. The cultures showed the bacillus coli communis which was probably a contamination. Dr. D. J. McCarthy and I carefully examined the muscular and nervous systems. There was no change visible to the naked eye in either the brain or cord. For microscopic examination the hemalum-eosin, Marchi, Weigert sheath, Nissl. and the fresh osmic acid methods were employed. The cranial nerves and the spinal cord were entirely normal on microscopic examination. The only changes found in the brain were small hemorrhagic areas in the region of the aqueduct of Sylvius. These were recent, without other evidence of inflammatory reaction, and may therefore be considered to have been either agonal or to have occurred a very short time before death. The muscles, Fig. I, the tongue and a bit of one of the pectorals, showed a distinctly pathological condition. The hemalum-eosin method revealed areas of small, round, nuclear infiltration betwen the fasciculi of muscle tissue, triangular or quadrangular in shape. The cells were of two types. The larger were small, round, deeply staining nuclei, one and one-half times the diameter of a red blood corpuscle, and strikingly resembled the cells of the thymus gland. They were, however, somewhat larger than the cells of a thymus of a fetus which was examined as a control. There were beside these, small round cells having nuclei of about the same size but with quite large cell bodies and staining a bright red with eosin. A few small round cells were seen scattered along the blood vessels in the reticular tissues. The cellular accumulations in the tongue were similar to those in the other muscle, except that the nodes were not so large. The types of cell appeared to

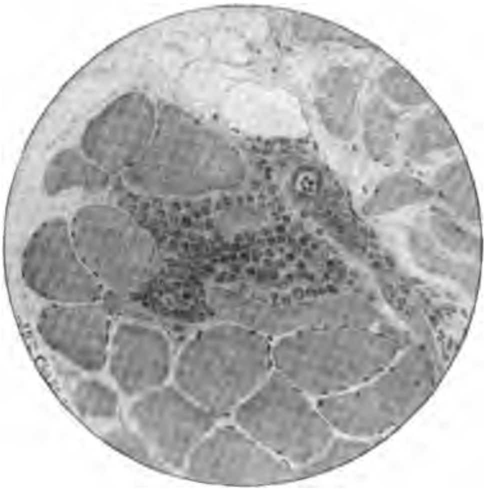

Fig. 1.

be the same. To sum up the lesions found were perivascular accumulations of free red blood cells in the region of the red nucleus and a lymphoid infiltration of the muscles.

In January, 1901, I published with Dr. D. J. McCarthy[1] the report of a case of asthenic bulbar palsy in which the thymus gland was persistent. I failed to appreciate at that time that there could be any relation between the thymus gland and asthenic palsy and regarded the pathologic finding as a mere coincidence without significance. Weigert was the first to point out that there might be a connection between the thymus gland, lymphoid infiltration of muscles, and myasthenia. In 1901[2] he reported a case with sarcoma arising almost surely from the remnant of the thymus and accompanied with an infiltration of the muscles by lymphoid cells. Later Link[3] reported lymphoid infiltration in the muscles with a persistent thymus. Hoedelmoser[4] reported a case with persistent thymus. Recently Henry Hun[5] has published an elaborate account of a case in which there was a lymphosarcoma of the thymus with the same muscular infiltration described by Weigert. He also gives an excellent bibliography. Goldflam[6] found a sarcoma of the lung the origin of which may have been in the thymus gland, and lymphoid infiltration of the muscles. In Oppenheim's[7] case, which seems to have been one of myasthenia gravis, there was a sarcoma of the anterior mediastinum.

The morbid anatomy and pathology of myasthenia gravis remain to be discovered, but till now the most frequent lesions found have been a persistent, or persistent and diseased, thymus and lymphoid infiltration of the muscles. The changes which have been found in the nervous system, slight lesions of the cranial nerves and their nuclei, microscopic hemorrhages in the floor of the fourth ventricle, and congenital abnormalities of the cord are not sufficient to explain the symptoms.

[1] The American Journal of the Medical Sciences, January, 1901.
[2] Neurologisches Centralblatt, Band XX., p. 597, 1901.
[3] Deutsche Zeitschrift für Nervenheilkunde, Band XXIII., p. 114, 1901.
[4] Zeitschrift für Heilkunde, Band XXXIII, p. 279, 1902.
[5] Albany Medical Annals, January, 1904.
[6] Neurologisches Centralblatt, Band XXI., p. 97, 1902.
[7] "Die Myasthenische Paralyse," Berlin, 1901.

ADDITIONAL CONTRIBUTION TO CASES OF MULTIPLE SCLEROSIS WITH AUTOPSIES.

By S. G. Webber, M.D.,
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In the July number of this Journal Drs. Spiller and Camp mention the rarity of multiple sclerosis in America and say that only six autopsies have been reported in American medical journals. Reference is made to the article by Drs. E. W. Taylor and J. W. Meyer in the Boston Medical and Surgical Journal of April 9, 1903, wherein is mentioned the rarity of the disease in Boston as compared with New York, and its much greater frequency in Europe than in America, also reference is made to the difficulty in many cases of making a correct diagnosis.

Prof. Dr. Dinkler[1] has lately published an article in which he especially mentioned the difficulty of diagnosis. He reports a case, which during life seemed to be one of transverse lesion of the spinal cord: but the autopsy showed it to be a case of multiple sclerosis.

This difficulty of diagnosis, especially in the earlier stages of the disease, may in part explain its apparent rarity. Patients attend a dispensary when the diagnosis is uncertain, hence the disease is not recognized. When more advanced they are not seen, because by that time their condition is considered hopeless and a physician is sent for only in some emergency, therefore their cases are never reported.

I have seen in private practice over twenty cases which have been diagnosticated as sclerosis; of these about half or a little more were marked with a question; the others were sufficiently well-defined and the diagnosis need not be doubted.

Two cases, in which I obtained autopsies, were diagnosticated as other than sclerosis—one as tumor of brain, but later its true nature was recognized; the other I thought was locomotor ataxia. As the patient died with obscure abdominal symptoms an autopsy was secured; appendicitis was found and the locomotor ataxia was discovered to be multiple sclerosis.

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[1] Deutsche Zeitshrift für Nervenheilkunde, XXVI., p. 233.

Another case was sent me many years ago as a case of chorea in an adult, by a physician very well posted in nervous affections; which proved to be multiple sclerosis.

I offer these two cases for publication because they add two to the number of autopsies, and because of the mistakes I made in diagnosis, as illustrative of the protean forms of multiple sclerosis, especially in the earlier stages.

Case I. Mrs. W., aged 22 years, had always been far from robust, but never feeble; she seldom lost a day at school, even when feeling poorly; she studied hard and stood well. Catamenia appeared at 12, she left school at 14; was a shop girl three years, standing all the time in the store. At this time she began to suffer from "faint spells" or fits, attended with some convulsive action. During these three years she began to have pain at the menstrual periods. Later she was cashier and could sit more and then had fewer of the faint spells.

About a year before I saw her she had trouble in the right ear, was sick about a week, when there was a discharge from the ear; she did not recover from this for two or three weeks. Before and after this disturbance she had slight headaches and dizzy turns in the morning. She made light of these and of all her symptoms. About three months before I saw her the dizziness was much more severe, and at time of visit was occasionally troublesome. Headache during these three months was once very severe, about that time vomiting.

There had been numbness of left thumb and leg, which, when touched, gave a sensation as if the hand touching it were covered with a glove. Weakness of left arm and leg. Ptosis of right lid, which was only temporary.

When seen the pupils acted well to light, the right rather less than the left; the left was the larger. Left internal strabismus was present and the left eye moved with a jerky motion in following the finger and was not abducted as far as the right. Vision not quite so clear in the left eye, double vision to left of median line was present.

Motion of face on the left side was somewhat diminished, the forehead wrinkled more on the right side; both eyes opened and closed naturally. She walked with an uncertain gait, especially with eyes shut; she could not stand with eyes shut and feet close together, and inclined to fall to the right.

Sensation was diminished on the left side of the face and on the left hand. She could not always tie strings of her skirt readily with the left hand. Swallowing and respiration were normal, and there was no sighing nor singultus. Pulse was 90 and weak.

Ophthalmoscope showed the right nerve rather red, veins large in both eyes.

These symptoms, headache, dizziness, vomiting, impairment of motion and sensation of the whole left side, seemed to justify diagnosis of tumor cerebri. A certain amount of nystagmus of the left eye when the eye was turned to one side was the only symptom strongly suggesting sclerosis, and this might well have been owing to the defective action of muscles caused by tumor.

Dr. Rudolf Finkelnburg[2] reports among cerebral tumors a case which was at first supposed to have been tumor of the cerebellum; but later the characteristic symptoms of multiple sclerosis appeared. No autopsy was obtained.

Mrs. W. was seen about eighteen months later. Her condition had changed; a part of the time she could not walk on the street, as her feet failed her. Tremor of the arms and head had appeared and was most severe when she moved. Her pronunciation had become jerky, so that speech was interfered with. The symptoms had developed rapidly, with many changes. She could not lie on the left side on account of dizziness. The dates of the different changes could not be obtained.

When seen the eyes were normal in motion, there was a sense of pressure in the head, but no headache. The hand trembled violently in any effort at motion, and also when lying at rest. She was in bed, and seeing me excited her so much that the tremor affected the whole body, which was greatly shaken; the legs were not affected while she was in bed so much as when she sat up. The control of the right leg was less than of the left. Sensation was not impaired to any extent.

Mental change was very marked; she was silly, made foolish remarks, entirely out of keeping with her character.

Her condition varied, at times some symptoms were better and again they became more marked. Deglutition was at one time very difficult. The tremor diminished somewhat, speech was less interfered with and became decidedly stacado. The legs were almost powerless; the right was at times drawn up and the reflexes were more active in it than in the opposite limb. Nystagmus was very marked. A bedsore formed over the sacrum; she had incontinence of urine. Her mind was more affected, and in the course of another year, about two and one-half years after she was first seen, she died.

Autopsy was twenty-four hours post-mortem. Only brain and spinal cord were examined.

Brain was comparatively small, but the sulci were not open. The brain seemed to have shrunken en masse, the arachnoid was filled with serum. Scattered throughout the cerebrum and cerebellum in the white substance were numerous patches of sclerosis, varying from a minute point to the size of a bean. These were most numerous on the right side. The right corpus striatum and the right corpus dentatum were much changed. Several sclerotic patches were in the pons, more on the right, and some were on the floor of the fourth ventricle and around the aqueduct of Sylvius. There were some spots in the medulla, one spot in the cord about an inch below the medulla. This is the note made at the time of the autopsy. I believe a few spots were found later on section of the cord, which did not reach the surface. The cord was much less affected than the brain, and both were very firm in texture.

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[2] Deutsche Zeitshrift für Nervenheilkunde 21, 1901-2, p. 446.

Case II. Mr. T., aged 44, elevator manufacturer for the previous six or seven years; worked hard in damp and cold places in new buildings. Eight or ten years before he came to me he fell ten or twelve feet, striking on his head and shoulders. He recovered from the fall. Eighteen months before I saw him it was an effort for him to move, he had no pain, no fever, was not confined to the house, was languid. This condition continued for about two months, then he gradually improved and felt well for about a year. Both feet then felt numb, he had no pain, but a disagreeable sensation, somewhat like a "sleepy" feeling, but less strong. This paresthesia extended to the knees, after four or five weeks to the thighs, and later suddenly was felt in abdomen, first in lower part and in a few days in the epigastrium. The sensation varied in intensity, was strongest in the feet, knees and lower part of abdomen and less between. He had a "felty" feeling in the soles of the feet and a slight girdle sensation, but no pain and no tenderness. It was an effort for him to walk straight; he had not full control of his limbs; a little loss of coördination.

Some years before his visit to me his eyesight was bad, vision was blurred, and for several days he was blind. He never recovered perfect vision. He did not have diplopia nor hemiopia.

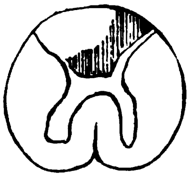
He had little difficulty in breathing when lying on his back and had to lie on his side to breathe freely.

He had no trouble in his hands, except a slight incoördination in the use of the left hand. Pupils, eyes and face moved naturally and well. He walked and stood poorly with eyes shut. Examination of the eyes with ophthalmoscope was difficult on account of spasmodic closure of the lids. The right fundus was normal; the left was not seen, as the eyes moved too much, possibly nystagmus was present. No excess of reflex action was seen. The record does not state what reflexes were examined. Sensation seemed unimpaired.

About six weeks from the above examination he died from appendicitis. The autopsy was made nearly forty-eight hours post-mortem. The appendix was ulcerated and there was general peritonitis.

The spinal cord was injured in two places in removing it, elsewhere it was firm. In the dorsal region the posterior columns were rather translucent and at the lower part of that region was a very well marked spot of sclerosis affecting the left side the most. In the lumbar region was a slight increase of translucency in the posterior columns. In no section was the anterior or lateral column affected.

In the medulla was a spot in the lower part of the left olivary body, and another in the vicinity of the upper part of the right olivary body. In the pons on the right side near the center was a spot small on the surface, much larger internally. In the left corpus striatum were a small and a large spot. In the hippocampus major on both sides were spots, more on the left. In the white substance of the corona radiata were a few small spots, chiefly on the left side. None were found in the cerebellum. On the right side the gray substance of the anterior corpus quadrigeminum was of a deeper color than usual, and this extended farther inwards on the left. No other change was found in the optic tracts.



The figure is from the spot of sclerosis in the dorsal region and is the only sketch made at the time of the autopsy.

When I examined Mr. T., I thought he was probably suffering from locomotor ataxia. The symptoms were not clearly indicative of that disease, nor were they the usual symptoms of multiple sclerosis. The explanation of this is found in the fact that the patches of sclerosis were in the posterior columns of the cord, while those in the brain were not numerous nor large enough to give characteristic symptoms. Had he lived longer probably the disease would have developed so as to clear up the diagnosis. I was fortunate in seeing the patient early in the disease, so that I had a chance to notice the early symptoms and observe them when they were not well defined, at a time when it was easy to mistake their significance.

Society Proceedings

PHILADELPHIA NEUROLOGICAL SOCIETY.

November 22, 1904.

The President, Dr. C. S. Potts, in the Chair.

A Case of Arteriosclerosis of the Nervous System.—This patient was exhibited by Dr. William C. Pickett. The man is sixty-seven years of age. Two years ago he began to be troubled with dizziness and headache; from that time he has slowly become "stiff all over," until now his walking is very difficult. Dr. Pickett had repeatedly questioned the family, and they insisted that at no time was there a sudden accession of these symptoms. The right leg and arm are decidedly more spastic than the left. Hemorrhage or thrombosis might be thought of, but the family insist that the symptoms have been gradual in onset. The knee-jerk is spastic and is greatest on the right side. Spasticity is much less in the arms and hands; but numbness of the fingers is complained of. The mental condition of the man is not impaired; his pupils react to light; bowels and bladder are well controlled; there are no sensory changes. Speech has become mumbling. It might be said that this is an ordinary case of senility, but Dr. Pickett thought we do not find such a history very often, and such cases have a new interest in the light of the somewhat modern conception of arteriosclerosis of the central nervous system. It seems to him that this case is not one of softening in the grosser sense, but must be one of arteriosclerosis, causing ischemia and impaired nutrition of areas, which, if thrombosis had occurred, would be the seat of softening, but in this slower process are the seat of degeneration and sclerosis. In the insane department of the Philadelphia Hospital, within a year, there have been two cases which might have been called simple demented paresis, but were different from any cases Dr. Pickett had ever seen. The history was that of a slowly increasing general spasticity with mental failure, and in one case, at autopsy, there was such a degree of sclerosis and atrophy of the frontal lobes—the prefrontal and anterior central gyri—that the contrast with the part of the brain back of them made a shelf-like projection. The explanation clinically and at autopsy was found in the arteriosclerotic brain-degeneration of Alzheimer. The arteriosclerosis of the larger vessels of the brain, the anterior cerebral, etc., was very striking.

Dr. Spiller thought that arteriosclerosis does produce great change in the brain in many cases, causing just such changes as in Dr. Pickett's case, and he said it is important to recognize them.

Dr. C. L. Allen thought that in most institutions where there are many old people, especially senile insane, in a certain proportion there will be symptoms incident to arteriosclerosis. He stated that he had recently had two cases of senile dementia, a man and a woman, presenting the picture of spastic paraplegia. The man died, and autopsy was denied, but he had arteriosclerosis, and the symptoms were probably due to arteriosclerotic changes in the spinal cord. The woman is still living, is bedridden, and has contractures of legs and arms. Before her symptoms became so marked she had increased reflexes and spasticity, which became greater and greater, until now the legs and arms are in a state of contracture. He regarded these cases as arteriosclerosis of the spinal cord.

He thought that many cases of vertigo and epilepsy could be explained by arteriosclerotic changes, and most of these cases, if they come to autopsy, show considerable arteriosclerosis.

Dr. Gordon thought that Dr. Pickett's case was probably one of arteriosclerosis. He cited two cases of his own in which he attributed the symptoms to arteriosclerosis. One of the cases had loss of reflexes, and the other loss of reflexes on one side and exaggeration on the opposite side, and still the arterial changes were uniformly distributed in both cases. He thought it hard to understand why, in some cases, there will be involvement of the posterior columns, and in others of the lateral tracts, while the arterial changes are equally distributed.

Hemiplegia Following Typhoid Fever.—This case was reported by Dr. C. S. Potts. F. L., aged twenty-eight years, family history good, previous history good. He had typhoid fever four years ago, and while convalescing became suddenly paralyzed on the right side. He was out of his mind three weeks immediately following. Speech was affected for ten weeks. When he came to the dispensary he had a right-sided hemiplegia, increased reflexes, Babinski's sign, ankle clonus, and right-sided paralysis of the face.

Dr. Potts thought the cause was either apoplexy or encephalitis, the three weeks of delirium being in favor of the latter.

Dr. Camp, in discussing Dr. Potts' case, exhibited a case from Dr. Spiller's service at the Polyclinic Hospital, which he thought might have sufficient connections with Dr. Potts' case to be of interest. The patient is twelve years of age. His family history is negative, and his previous medical history contains nothing of importance. He had scarlet fever at seven years of age, and had previous attacks of chorea. On May 4 of the present year he developed attacks of chorea. No blood examination was made, but the symptoms were typical. On May 19 he stopped using the right arm and leg, and could not lift them from the bed. At the same time they became the seat of irregular jerky movements. The doctor in attendance was not sure that the face was involved. On May 29 he developed a severe attack of pneumonia, which had its crisis on the 6th of June. He began to use the right arm normally as soon as he sat up in bed after the pneumonia. He was much emaciated and went to the country. On his return, seven weeks ago, he seemed perfectly healthy. Two weeks ago his mother noticed that he began to drop things from his right hand, and would not use the hand. He also became peevish and irritable.

When examined, November 16, 1904, it was noticed that the right side of the face was weaker than the left, although the upper part of each side of the face was normal. The tongue deviated to the right when protruded. Saliva dribbled from the left corner of his mouth. His knee-jerks were normal on each side. There was no Babinski reflex, although there was a tendency toward permanent hyperextension of the toes. Oppenheim's reflex was not obtained. Choreiform movements of the right arm and leg were distinct. He was given Fowler's solution, gtt. iii. tid. The choreiform movements seem now to involve also the face. The electrical reactions are normal. There is no atrophy. The diagnosis of his condition last May rests between hemiplegia developing during typhoid, or typhoid fever with a coincident attack of chorea. The condition at present may be a weakness remaining from the hemiplegia and now complicated by chorea, or it may be a case of paralytic chorea. There is also another possibility, there may be a lesion in one of the superior cerebellar peduncles, which, as is well known, will produce choreiform movements of the affected side.

Dr. Potts added that his patient had recovered the power of speech, and is now mentally bright. He said that L. Stein (*Pest. Med. Chir. Presse,* Jan. 10, 1904, p. 44) had recently reported a case of hemiplegia occurring during typhoid fever, and had commented upon the rarity of this sequel.

Dr. Gordon said that he had recorded, last year, a case of hemiplegia occurring during convalescence from typhoid fever. The symptoms came on suddenly, but the patient recovered entirely. These disturbances occurring in the last period of typhoid fever are only transient in character, according to the literature and his own observation.

Dr. Camp added that his case now presents the symptoms of a paralytic chorea, hemiplegic in distribution. It is rare to have the face involved in such cases. In the present case the face had been distinctly involved when first seen, which, if it is a case of paralytic chorea, marks it an exceeding rare one. On a careful review of the literature, published in the June number of the *University Medical Bulletin,* he was unable to find a case of paralytic chorea in which the face was involved.

A Case in which the First Left Temporal Convolution was Destroyed in an Adult Without Causing Word Deafness.—This case was reported by Dr. W. G. Spiller and Dr. H. B. Allyn. The patient, a man fifty-seven years of age, right-handed, had had two apoplectiform attacks, one in 1881 and another in 1886. Notwithstanding these attacks he could understand all that was said to him; could relate anecdotes and showed very few symptoms of aphasia in his later life. He was a man of considerable mental ability. At autopsy it was found that there was a complete destruction of the first left temporal convolution. The second was well developed. We must assume that the retention of function of word hearing was due to the preservation of the second left temporal convolution, or to the unusual development of the first right temporal convolution. A very similar case has recently been reported by C. S. Freund.

Dr. Mills thought an interesting point in connection with the specimen exhibited was that there appeared to be no arrest of development of the hinder part of the third frontal convolution, which was apparently of normal size. He thought some explanation might be reached indirectly from this fact if the man were a right-handed individual, as it appeared he was. He stated that in a case of his own, and in other cases reported of word deafness, after the lapse of years secondary involution of the third frontal convolution and other parts concerned in the mechanism of speech was present. He thought it looked as though this man's word-hearing center might be on the right side, rather than on the left, where it usually is in right-handed persons. He stated it as his belief that the lower portion of the hinder part of the first temporal convolution and the upper part of the second temporal convolution are concerned in word-hearing. He thought the explanation of this case lay in the unusual development of both temporal regions.

Dr. Dercum cited a case of Bramwell's in which there had been an attack of motor aphasia, but in which a short time subsequently the man spoke as well as before. At the autopsy it was found that there was a complete destruction of the third frontal convolution of the left side. Here was a case in which the right side of the brain had been functionally active as well as the left. He thought it possible that in persons unusually gifted there may be an education of both centers; that is, such persons would have ambidextrous or, more properly speaking, ambisinistral brains.

Dr. Spiller stated that the view brought forward by Dr. Dercum was Freund's explanation of his case, that the word-hearing center had been latent on the right side, and was called forth when unusual demands were made upon the brain. Referring again to the case reported by Dr. Allyn and himself, Dr. Spiller said that he had asked Dr. Allyn particularly about the man's appreciation of music, because so little is known about the center concerned in the hearing of music. The patient had not been musical. The anterior part of the first left temporal convolution is supposed by some to be the seat of music hearing, while the center for word hearing is in the posterior part of this same convolution.

A Case of Multiple Spinal Sclerosis, with Remarks Upon the Pathogenesis of the Disease.—This paper was read by Dr. F. X. Dercum and Dr. Alfred Gordon.

The Earliest Cases of Disseminated Sclerosis with Necropsy and Microscopical Examination Reported in America.—Dr. Charles K. Mills thought it would be well, from a historical standpoint, to refer to the fact that the first cases of multiple sclerosis with necropsy were recorded by E. C. Seguin, of New York, in 1878; and that he (Dr. Mills) in 1879 reported a case with necropsy and microscopical findings in the *Hospital Gazette* of New York. As the cases both of Dr. Seguin and himself were apparently not well known to American neurologists, it might be well to call attention to them in some detail.

The notes of Dr. Seguin were on two cases, which were recorded in a paper which was entitled: A Contribution to the Pathological Anatomy of Disseminated Cerebrospinal Sclerosis, this having been presented at a meeting of the New York Neurological Society, held February, 1878. The paper was published in the JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 5, April, 1878.

The first case was that of a man twenty-nine years old. His first symptoms were disturbance of vision and incoordination after marching. In less than a year his symptoms were well marked. The case is well recorded, although not as regards studies in sensation, reflexes, coordination, etc., exactly after the manner in which a record of to-day would be made. Examination of the reported facts, however, show that the patient had a spastic, paretic gait, incoordination both in the lower and upper extremities, intention or action tremor, nystagmus, irregular disturbance of the sensation, exaggerated reflexes, disorder of speech, and other symptoms clearly pointing to disseminated sclerosis. The man died a little more than six years after the first symptoms were observed.

The second case was that of a young woman, twenty-three years old. She first began to suffer with weakness of the right leg, which developed into a marked paresis, sensibility to pain being a little dulled in the leg and foot. Dr. Seguin first believed that this case was a hysterical disorder, a significant fact when it is remembered how often hysteria and disseminated sclerosis have been confused. About six months after her first symptoms she somewhat rapidly became paraplegic, her hands also showing some paresis. Later bed-sores developed, apparently, according to the reporter, from want of care while under treatment in a hydrotherapeutic establishment. The autopsy was made by Dr. Seguin, the spinal cord only being removed. This second case was not nearly so well recorded clinically as the first.

The brain and spinal cord in Case I were hardened in potassium bichromate; sclerosis in patches was found in various parts of the cerebrum, caudatum and in the oblongata. The exact position and extent of the foci of sclerosis are given in numerous instances. Sections of the spinal cord were made from the cervical, dorsal and lumbar regions, these being stained with carmine and hematosin. The foci of sclerosis in various parts of the cord are described.

In the second case the microscopical examination was only of the cord, and Seguin gives a description sufficiently full to show that this, like the first case, was clearly one of disseminated sclerosis. In summarizing the cases he remarks that the sclerosis nodules were of various sizes, and occupied the most diverse regions of the cerebrospinal system, involving the gray as well as the white matter. He makes a differential study of the histological appearance, as observed in an early stage, in one in which the morbid process has considerably advanced, and in a third in which the most extensive changes are to be seen. Descriptions are given of the appearance presented by the neuroglia, nerve cells, vessels, etc., and a plate, including sixteen illustrations, is given. The histological descriptions are good for the time when the investigation was made.

It will be remembered that Seguin had pursued his clinicopathological studies in the wards and laboratory of Charcot, and also later with Forel. Certainly the record of these cases should not be overlooked in speaking of the cases of disseminated sclerosis recorded in this country, even though it is true that the more recent work of Spiller and others regarding the same disease has been of a more exact and elaborate kind.

The case reported by Dr. Mills in 1879 was one which had been carefully studied clinically and one in which a necropsy had been obtained and a subsequent microscopical investigation made. The case proved to be one of disseminated sclerosis with diffuse, and in some places coalescing patches in the dorsal and lateral columns. This case had not attracted attention in the résumé of the literature of disseminated sclerosis and combined sclerosis, probably in the main because of the place of its publication. It was published in consecutive numbers of the *Hospital Gazette of New York,* Nov. 22 and Nov. 29, 1879. The journal was chiefly devoted to the publication of clinical lectures and other clinical material, and while its circulation was large, it had only a brief existence, and probably is seldom consulted by neurologists and writers on neurological topics.

The patient was a woman, thirty years old, whose past history was uncertain, but who said that the first symptoms of her disease came on about a year before her admission to the hospital after an attack of typhoid fever. It seemed probable that the invasion of her disorder was earlier than the time fixed by the patient. The dominating features of the case were spasmodic paresis and ataxia, both lower and upper extremities being highly spastic; knee-jerks and other deep reflexes were exaggerated; intention tremor was shown when the patient attempted to walk; it was also shown in her efforts to use her upper extremities. Her speech was distinctly of the scanning and syllabic character; she showed some mental hebetude and deficiency; partial atrophy of both optic nerves was present but nystagmus was absent. The changing emotional states which accompany disseminated disease of the pons and oblongata were particularly marked. Impairment of sensibility, especially in the lower extremities, was determined by careful investigation. When most of the examinations were made the ataxic symptoms were largely masked by the spastic manifestations. During the two months preceding her death she grew rapidly worse. The spasticity markedly increased, she became more and more helpless, and was soon totally bed-ridden. The complaints of pain, especially in and about the knees and elbows, became more frequent, and now and then she would have terrific stabs of pain in her legs, thighs, arms or trunk. Her speech and mental strength failed steadily, involuntary evacuations began two weeks before her death. In the last five days of her life her knees and ankles were red, hot and swollen, like those of a person suffering from acute inflammatory rheumatism. A sacral bedsore formed, and other similar sores at the ankles and on the feet. Both brain and cord were examined carefully in the fresh state and after hardening. Besides the appearances of a chronic disseminated sclerosis, the microscope revealed evidences of a recent acute or subacute inflammatory affection of both the cord and brain. Sclerotic nodules were described as occurring in both thalami, in the crura, pons and oblongata. The lumbar enlargement and lower portion of the thoracic cord presented a shrunken and atrophied appearance even to the naked eye. The results of the microscopical examination of the cord, oblongata, basal ganglia and cerebral hemispheres are given, these showing typical advanced disseminated sclerosis. The extent and position of the sclerotic areas are described in detail.

Dr. Camp referred to two cases of multiple sclerosis with autopsy, reported by Dr. Spiller and himself. He stated that in these cases the incontinence of urine and feces was inconstant, while in Dr. Dercum and Dr. Gordon's case they were persistent, and that this was probably due to the more advanced stage of the disease. In both of the cases Dr. Spiller and he had reported symmetry of the pathological lesions was a marked feature, which was different from the findings mentioned in Dr. Dercum and Dr. Gordon's case. As regards the theory of the vascular origin of the disease Dr. Spiller and he had found the blood vessels in the sclerotic areas much thickened, but outside these areas they were practically normal, which would point to at least some connection between the two.

Dr. Dercum thought the facts were not such as to justify the belief that this disease is due to developmental defect. On the other hand he did not think it had its origin in the nervous elements or blood vessels. We are driven to the conclusion that the disease begins in and remains most pronounced in the glia. Its origin is still a matter for speculation.

Dr. Spiller said that in one of the early cases under his care there was secondary deegneration and ophthalmoplegia. During life the diagnosis of multiple sclerosis was made. He called attention to another view held in regard to the nature of this disease, namely, congenital malformation of the spinal cord and brain. Exposure to cold and damp is also important. This had been borne out in his own experience, as there was a history of exposure to either cold or dampness in every case he had had. The case reported by Drs. Dercum and Gordon had entered the hospital under Dr. Spiller's care, and the diagnosis of multiple sclerosis had then been made. Later the case had been in the service of Dr. Potts.

Dr. Gordon stated that he was familiar with the influence of damp and cold upon this disease. He stated that there was a suspicion of specific disease in the case they had reported. The woman had never had living children, had several miscarriages, and cystic ovaries. There was also a history of trauma. They, therefore, had no solid basis as to formation of an opinion upon the etiology of multiple sclerosis, as multiple factors enter into the etiology of the affection.

A Case of Tic Convulsif (?) in a girl of Six, and a Case of Hemiparesis, Muscular Rigidity and Exophthalmus with Von Graefe's Sign.—These cases were reported by Dr. Joseph Sailer.

Dr. Gordon said that although Dr. Sailer had stated there was no hysterical stigmata, he did not think that excluded the possibility of the case being one of hysteria. The typical symptoms of Gilles de la Tourette's disease were wanting, particularly echolalia and coprolalia. The complete disappearance of the symptoms under the influence of a small dose of a drug, speaks very much in favor of hysteria.

Referring to Dr. Sailer's second case, with regard to the presence of Von Graefe's sign in other conditions than exophthalmic goiter, Dr. Camp stated that he examined a case in Dr. Spiller's clinic in the Philadelphia Hospital, this fall, in which Von Graefe's sign was very distinct, and which was not a case of exophthalmic goiter.

Dr. Sailer called attention to the fact that he had been careful to put a question mark in the title of his first case, and that from a further study of the case he was inclined to believe it to be one of hysteria, but he thought even that diagnosis did not seem entirely satisfactory.

Specimens from a Case of Fracture of the Fifth Cervical Vertebra, Causing Total Transverse Lesion of the Cord.—These were exhibited by Dr. F. X. Dercum.

T. W., male; black; aged twenty-four; laborer; was admitted to the Nervous Wards of the Philadelphia Hospital, November 14, 1904. Clinical diagnosis, fracture of the fifth, sixth and seventh vertebræ, with hematomyelia of the same region. Chief complaint, paralysis of trunk and limbs, caused by an iron bucket falling on the head and neck. The family history is unimportant and negative, as is also the previous medical history. The patient was never ill in his life before. Has never had syphilis; thinks he had gonorrhea once when very young. Has used alcohol and tobacco very moderately.

History of Present Illness.—About 4 P.M. of July 13, 1904, the patient was engaged in pulling up buckets of cement to a height of seven stories, each bucket weighing from seventy-five to one hundred pounds. He was struck on the back of the head and neck by the bottom of a bucket, which had descended suddenly and very rapidly. He immediately became paralyzed over his entire body. Control of the bowels and bladder was lost at the same time. He was taken to the Medico-Chirurgical Hospital, and remained there until his transfer to the Philadelphia General Hospital. He was, according to his own statement, entirely conscious at the time of the accident. Shortly after, however, he became delirious, and remained so about three days. At the Medico-Chirurgical Hospital a loss of sensation was noted, involving the trunk from the nipples down anteriorly and posteriorly from the lower border of the scapulæ. This loss was total and included all forms of sensation. Over the arms there was an absence of sensation extending from the elbows to the ends of the fingers. There was also incontinence of urine and feces. An X-ray examination revealed a fracture involving the fifth, sixth and seventh cervical vertebræ. There was no abnormality of any of the viscera. Patient was able to take food and to digest it well.

Notwithstanding the fact that he was placed upon a water-bed, a bedsore developed over the buttocks on July 27. By August 15, similar sores made their appearance over the heels and ankles, by September 1 over the heads of the fibulæ and elbows, and by October 10, two small bedsores over the left scapulæ appeared.

At the time of his admission to the Philadelphia General Hospital the patient complained of a sensation of cold, involving the entire body and all his extremities. The penis was in a state of priapism. Bedsores noted at the Medico-Chirurgical Hospital were also present on admission, and in addition were sores in both popliteal spaces and another over the right shoulder.

Physical Examination.—The patient was a well-nourished negro, of twenty-four. Mucous membranes were of good color. The skin over the malar bone had taken on a whitish color. Over the entire body, but more especially on the feet, legs and upper extremities, the skin was much thickened, hard, and showed evidence of desquamation. The head was of normal shape, and covered with an abundant supply of curly black hair. There was a depression of the skull just over the occipital prominence on the left side; this depression was not deep, but easily made out; the face did not show any asymmetry nor palsy; the patient was able to show his teeth, draw the corner of his mouth to each side, and the naso-labial folds were of equal prominence. The patient could talk; answered questions well; whistled and wrinkled his forehead. He had all of his teeth, which were in good condition. Deglutition was normal. The patient laid flat on his back, with his upper extremities lying across his chest, in a state of contracture. He was unable to move any part of his body, and if an attempt were made to raise any limb, or disturb him in any way, he seemed to suffer pain. His chest was well developed; respiratory excursion was good on each side. The lungs were resonant throughout; there were no adventitious sounds. Respiration was diaphragmatic; pulse was rapid, full and regular. The heart was normal in outline; the apex was at the fifth interspace in the mid-clavicular line; the mitral sound was normal; there were no murmurs. There was a slight accentuation of the second aortic sound.

Eyes.—The pupils were equal, reacted to light, accommodation and convergence rather promptly. No extra-ocular palsies were noted. The conjunctivæ were discolored yellow. The tongue was protruded promptly in median line; was clean, and slightly fissured.

Back.—On examining his spinal column, patient had to be turned very gently on his right side, and in going over spinal column, from below upwards, tenderness to pressure existed over the first dorsal vertebra, and thence upward over the cervical vertebræ, until the fourth was reached. This seemed by all means the most painful. No crepitus could be elicited. The patient was unable to turn his head; his neck seemed to be rigid.

Arms.—When an attempt was made to raise the arms, pain was experienced by the patient, and when they were raised they fell heavily down upon the chest. The arms were in a state of contracture. The patient was unable to perform any movement with the arms; the grip in each hand was gone. The skin over the hands was thick, hardened and desquamating. The reflexes were abolished. There seemed to be some atrophy. Sensation in upper extremities was lost up to the junction of the middle and upper thirds of the arm.

Legs.—The patient was unable to raise either limb from the bed, and when an attempt was made the disturbance gave pain. The patient was unable to perform any movement with the legs. The reflexes on each side were abolished. Irritation of the plantar surfaces did not give any response. The skin over the feet and toes was much thickened, hardened and desquamating, as over the hands. Each foot was markedly swollen, and somewhat edematous. Sensation in each extremity was lost. The penis was in a state of semi-priapism; the glans was swollen and excoriated.

Sensation in all forms was lost over the entire body anteriorly as far upward as a line drawn from shoulder to shoulder, and posteriorly by a line drawn from the upper border of one scapula to the other. Above this level, anteriorly, sensation was normal, but posteriorly there was marked hyperalgesia over the entire cervical region. Muscles of the trunk were paralyzed. Both sphincters were paralyzed.

Trophic Changes.—The entire body had a cold feeling, the skin was thickened and hardened over extremities as has been described. The nails were brittle and ridged. Bedsores were present as described. Unilateral sweating of forehead on right side was also noted.

November 7, 1904.—For last three or four weeks the patient had been complaining of sharp pains radiating from right ear down to lower part of the mouth, but only during mastication or yawning. The patient also stated that there was a "watering" from both eyes, but especially from the right eye. He complained of pain in upper part of the spine, between the shoulders, and radiating to both arms.

November 9, 1904.—To-day there was free perspiration on left side of body, covering face, neck, arms, forearms, hands and trunk.

November 10, 1904.—The patient was very restless and noisy during the night; he twitched over the entire body. He complained of difficult swallowing, and his respirations were labored. He also complained of pain of a severe character in his neck, and at times over his entire body.

November 10, 1904.—Eye examination by Dr. de Schweinitz. No change in optic discs, both of which are pysiologically cupped. The veins and arteries are about normal in size; the arteries are exceedingly pallid, the veins dark. Roughly tested the visual fields are normal in both eyes. The pupillary reactions are normal. No paralysis of any external ocular muscles.

November 12, 1904.—Patient had been expectorating a good deal of mucus. Examination of the lungs showed moist râles over the bases of the lungs. The temperature of patient had been rather sub-normal since yesterday.

November 14, 1904.—Patient died at 6.45 A.M.

Autopsy Records.—Pathological diagnosis, fracture of fifth cervical vertebræ. Acute cystitis; edema of lungs; cloudy swelling of kidneys.

Body of an adult, male, negro. Extremities considerably emaciated. Over both elbows, posteriorly are two ulcerated areas, about three inches long, by two wide, both communicate with the elbow joint, the surfaces of which are much roughened. Over both scapulæ are small areas of ulceration. Over the buttocks there is a bedsore about eight inches in diameter. On the outer surfaces of the tibia are ulcerrations and likewise over the external malleoli.

The peritoneum is smooth and glistening. There is a slight amount of clear yellow fluid. The appendix extends downward, and is free from adhesions.

The lungs are distended, and nearly meet in mid-line. Both pleuræ contain a slight amount of clear yellow fluid. There are a few easily broken adhesions at both apices. The left lung is pale in color, soft, crepitant, from cut surface pale frothy fluid escaped on pressure. The right lung, lower, middle and posterior portions of upper lobes, purplish in color, firm, non-crepitant; from cut surface exuded a large amount of slightly tinged fluid, free from air. A bronchial gland showed caseous degeneration.

The pericardium is smooth and glistening and contains about 50 c.c. of clear fluid. The heart muscle is very pale in color, but firm. Some fibrous change is noted at the tips of the papillary muscles. The endocardium is normal.

The spleen is enlarged, pale in color; capsule smooth and glistening; cut surface shows trabeculæ and bodies quite prominent.

Kidneys considerably enlarged, pale in color; capsule strips easily; cortex thickened, numerous red striæ present; pyramids much injected.

Bladder.—Mucous membrane dark in color, and shows numerous irregular areas of hemorrhage; in one or two places the mucous membrane is absent.

Liver very pale in color, cut surface shows some congestion of vessels. Common bile duct patulous.

Small intestine throughout its entire extent shows marked congestion. Large intestine shows a few areas of congestion.

The brain shows no other gross changes than marked anemia.

The spinal column shows a transverse fracture of the fifth cervical vertebra. Fragments displaced. Cord firmly compressed at this point.

It was probably the sixth segment of the cord that had been destroyed, though the lesion had probably invaded also the fifth. The fourth cervical segment had evidently escaped, thus permitting diaphragmatic respiration to continue so many months. The level of the anesthesia is about that which one would expect to find in injury of the sixth and fifth cervical segments. The level of the anesthesia noted at the Medico-Chirurgical Hospital, immediately after the accident, was lower than that subsequently noted by Dr. Dercum. Doubtless this is to be explained by extension of inflammatory and degenerative changes upward above the site of the original lesion.

Periscope

CENTRALBLATT FUER NERVENHEILKUNDE UND PSYCHIATRIE

(November, 1904.)

1. On the Prognosis of Acute Polioencephalitis Superior (Wernicke). W. Spielmeyer, Freiburg.

1. All chronic processes are to be excluded from Wernicke's disease and acute encephalitis constitutes it only when the eye-muscle region is the center of the lesion. Spielmeyer reports the nineteenth fatal case of this polioencephalitis superior acuta. The patient was an alcoholic, became delirious and then comatose. He had first external strabismus, then total ophthalmoplegia. He died after twelve days. At autopsy capillary hemorrhages were found beneath the aqueduct of Sylvius. Spielmeyer speaks of a *triad of symptoms;* the rapid ocular palsies, the severe involvement of the sensorium, and the ataxia in gait and speech. The duration in these cases is from two days to a month. The patients all have been drinkers, except Wernicke's first, who was a young girl poisoned by sulphuric acid. Most of them showed other effects of alcoholism. The lesion is different microscopically from cerebritis, in the absence of true inflammation. The newly formed vessels in the central gray matter are due to the alcoholism, and the hemorrhages are analogous to diathetic hemorrhages. Recovery or improvement in polioencephalitis superior has occurred twelve times. Only two cases have perfectly recovered, the other ten having a persistent ophthalmoplegia. Cases ascribed to influenza have never been verified at autopsy. The extent of the ophthalmoplegia has no significance in prognosis. The delirium and stupor must be counted among the cardinal symptoms, and the case is likely to be mistaken for simple delirium tremens. The mental and general physical state is most important. The rapid pulse often ascribed to lesion of the vagus nucleus is more likely due to degeneration of the heart muscle.

(December, 1904.)

1. Apratic Symptoms in a Case of Senile Dementia. Dr. H. Marcuse.
2. On Some Noteworthy Mimic Motions of the Hand. Dr. Ernst Jentsch.

1. *Apractic Symptoms in a Case of Senile Dementia.* Dr. Harry Marcuse, formerly Assistant Physician of the Berlin Asylum at Dalldorf, discusses the apraxia of Liepmann, which is the inability, in spite of an intact condition of the sensory and motor apparatus, to make voluntary movements according to purpose.

The patient was a woman, considerably demented, showing defective orientation in time, place and personality, together with transcortical motor aphasia. The defects of voluntary acts, Marcuse says, were midway between sensory and motor apraxia, and might be called amnestic apraxia: in Liepmann's phrase, it is a *derailing* of impulses. The cause in this case was not apoplexy nor any focal lesion, but senile atrophy of the brain.

2. *On Mimic Movements of the Hand.*—This is an attempt to assign a rational origin to various common gestures, such as snapping the fingers. Jentsch's paper is psychological.

Wm. Pickett (Philadelphia).

ARCHIV FUER PSYCHIATRIE UND NERVENKRANKHEITEN

(Vol. XXXVIII, 1904, No. 3.)

1. A Contribution to the Knowledge of the Cerebral Palsies of Children. H. Wachsmuth.

2. The Palsies of the Parturient Woman. R. von Hösslin.
3. A Cyclops Monster with Microcephaly and Microgyri. O. v. Leonova v. Lange.
4. Neuro-pathology and Psvchiatry. C. Förster.. .. ..
5. A Case of Carcinoma of the Pons and of the Right Temporal Lobe Secondary to Cancer of the Uterus. Kufs.
6. Recurrence of Electrical Sensibility of the Brain following Temporary Anemia. V. Scheven.
7. Functional Hemiathetosis. Seifert.
8. Spasmodic Torticollis. Steyerthal and Solger.
9. Examination of the Stigmata of Regeneration in 251 Insane Men. R. Ganter.
10. Experimental Lesions of the Central Nervous System in Chimpanzees. M. Bothmann.
11. Classification and Nomenclature of the Psychoses, with a Review of the Pretenses of the Physician's Examination. A. Hoche.
12. Reviews. 1. E. Hitzig. Physiological and Clinical Examination of the Brain. 2. E. Schultze. The Psvchoses of the Military Prisoners, with Suggestions for Reform. 3. W. His. The Development of the Human Brain during the First Month.

1. *Cerebral Palsies.*—Five cases are reported clinically, with the necropsy report in one case. The first case was idiocy and epilepsy combined with asymmetry of the head, the right side being the smaller. Arrested development of the brain, microgyria and thinning of the spinal cord were also present. The most important finding at the necropsy in this case was the relative difference between the weight of the cerebral hemispheres, the left weighing 580 g. and the right 260 g. The second case was one of idiocy and epilepsy with hemianopsia. The third case was one of idiocy and epilepsy with dwarfishness, a case of cerebral palsy with paralysis. The fourth case was one of right-sided paralysis and hemiatrophy with athetosis and without positive epilepsy. The fifth case was one of idiocy and epilepsy. The study of these five cases, together with those already reported by the author in Volume XXXIV, No. 3, leads the author to agree with Bourneville that the epilepsy in these cerebral palsies ceases during the fortieth and fiftieth year; and while there are no differences noted between epilepsy in idiocy and those of the cerebral palsies, as first observed by Koenig, there are differences noted between the epilepsy in the ceebral palsies and idiopathic epilepsy, as was originallv claimed by Wuillameir. Aura, stertor, froth, tongue bite, and particularly drowsiness subsequent to the attack, are very rare phenomena in the epilepsy of the cerebral palsies.

2. *Palsies of the Parturient Woman.*—This is an exhaustive inquiry into the varieties, the cause and the treatment of these palsies. A consideration of the palsies without pathological findings (hysteria and myasthenia gravis) are first taken up; then follow the cerebral palsies, (*a*) those caused by simple apoplexy; (*b*) those in albuminuria (eclampsia); (*c*) those from thrombosis of the brain; (*d*) those caused by cerebral emboli; and (*e*) those subsequent to other diseases of the brain, tumors, etc. Under heading three are described the impressions the spinal cord diseases make upon the child-bearing woman. The relations of tabes, disseminated sclerosis, myelitis, etc., are described in turn. The pathogenesis, pathological anatomy, symptomatology and therapy are taken up in detail. Under heading four is described the influence of the uterine centres upon conception, pregnancy and birth. The paper is lengthy, instructive and of considerable general, as well as technical, interest. The paper is unfinished.

3. *Cyclops Monster.*—The pathological findings were observed in a premature child (possibly 7 months) still born. The mother was well; had had seven children born living, three of which were then living. The conclusions the author reached after careful examination of the entire nervous

system of the fetus and comparison with the normal one are: (1) When a fibre system takes origin from a distinct centre but has not a defined terminal nucleus, or when the last is imperfectly developed, such a fibre system is marked out very distinctly, and may be highly developed, but never presents normal characteristics (usual number of fibers, etc.). (2) When a fibre system is thinned there is shrinkage of the gelatin ground substance in which the fibre system terminates as end bulbs, etc.) These results corroborate the findings in previous cases by the same author. An interesting microscopical finding in this case was the fusion of the orbits, although each eye was distinctly separate, *i. e.*, two lenses, two sclera, two retina, etc. The drawings presented are numerous and illuminating.

4. *Neuropathology and Psychiatry.*—This is a lecture delivered at the yearly meeting of the German alienists in Göttingen during April, 1904. A review of the work done is given and suggestions are made for work in the future.

5. *Carcinoma of the Pons.*—In 1898 Buchholtz was able to find 66 cases of carcinoma of the brain. Of that number 41 were metastatic. The patient was 60 years old, and died apparently from sepsis. The brain weight was 1170 g. Two tumors were observed in the brain; one of the size of a hazel nut was situated behind the corpora quadrigemina at the beginning of the fourth ventricle. The second tumor, comparatively hard, was situated on the right temporal lobe, mid portion (gyrus hippocampus and gyrus fusiformis). There was a tumor about the size of a walnut within the wall of the uterus. The body of the uterus was free. The section was stained in hematoylin-eosin, alum-carmine, van Gieson stain, and Wolter stain for the fibers. In all the preparations the characteristic appearances of carcinoma were seen.

6. *Electrical Sensibility of the Brain.*—The experiments were carried out in rabbits. Thirty-three investigations in all were made. Rabbits were selected, as monkeys and cats were found by Mott and Hill in their experiments on the physiological and anatomical changes in the cerebral cortex resulting from anemia produced by ligation of the cerebral arteries, to die, usually within 24 hours afteh the ligation. In dogs the collateral circulation was too rapidly carried out to give satisfactory results. The faradic current was employed. Failure of response to the current was noted usually within 2—4 minutes after ligation, and occasionally as late as 5—7 minutes. In general, it was found that when the compression was not for a longer period than 10—15 minutes the return of irritability of the brain was very prompt, usually within 2—5 minutes after the withdrawal of the ligature. When the time of compression was increased the return of irritability was correspondingly low. When the compression was persisted in for 25—30 minutes the full return of irritability was very slow, four and one-half hours being required in one case. After thirty minutes' compression the animals usually died, so that no positive results could be obtained. The experiments show the great resistance of the brain to insults, and also its recuperative power.

7. *Functional Hemiathetosis.*—This, the author states is the first of its kind reported. The patient was a shoemaker, 37 years old, who complained for about four weeks of coldness and numbness in his right thumb and radial side of his right forearm. Within a short time the entire hand became affected and he could no longer use the hammer at his work. Athetoid movements were observed in the fingers of the right hand. No hysterical stigmata were noted. The patient was hypnotized and given verbal suggestion. After two treatments he was practically cured and returned to his work within a month.

8. *Spasmodic Torticollis.*—Three cases are added to the literature, which is carefully reviewed. As the author pertinently remarks, an ana-

tomical examination of a case of wry neck has not yet been made, and much depends upon what such an examination should disclose.

9. *Stigmata of Degeneration.*—This article is practically the continuation of a former article by the author on the same lines. In this paper the color and flecks on the iris, the ear, palate, alveoli, teeth, skeleton and hair are considered. With the exception of red and violet, all the colors of the rainbow are represented. Bluish-gray was observed most frequently (99 cases), and light-blue the next (77 cases). Flecks or points on the iris were present in 88 cases, or 35 per cent. Defects of the ear were lacking in only 6 per cent, a single anomaly of the ear was present in 8 per cent., two anomalies in 17 per cent., 3 anomalies in 19 per cent., four anomalies in 22 per cent., five anomalies in 13 per cent., six anomalies in 9 per cent., seven anomalies in 2 per cent., eight anomalies in 3 per cent., and nine anomalies in one individual. Defects of the palate were present in 71 per cent. of cases, anomalies of the alveoli and palate in 23 per cent. Defects of the teeth were found as follows: Anomalies in the interval between the teeth, 12 per cent; (*b*) positive anomalies, 45 per cent.; (*c*) changes in form, 63 per cent. Anomalies of the skeleton were present in 53 per cent. Abnormal hairy sites were present in 15 per cent. The mental affections of the 251 cases were imbecility in 80, dementia præcox in 60, paresis in 24, paranoia in 37, epilepsy in 16, dementia sec. in 12, senile dementia in 12, and other forms in 10.

10. *Experimental Lesions.*—This arbeit was carried out under the supervision of H. Munk, and is indicative of the careful, long-drawn-out experiments of the investigator. The article cannot be abstracted within a short space.

11. *Classification of Psychoses.*—The author does not propose a new classification or nomenclature, but criticizes judiciously the present trend toward complexity. The diagnosis he states is the most important item.

12. *Reviews.*

A. F. WITMER (New York).

REVUE DE PSYCHIATRIE ET DE PSYCHOLOGIE EXPERIMENTALE

(July, 1904.)

1. Dementia Associated with Circumscribed Lesions of the Brain. VIGOUROUX.
2. Psychological Study of Stereotypy. DROMARD.

1. *Dementia and Cerebral Lesions.*—It has been observed in cases of circumscribed brain lesions that in some cases there is following dementia, while in other cases of lesions of the same size and location the mental faculties are preserved. The dementia which follows this class of cases is supposed by the author to be due to a more or less diffuse process spreading from the central lesion and invading the surrounding cortical territory, destroying association tracts. These diffuse changes are inflammatory and infectious in character, and more often and more readily follow when the lesion has occurred in a brain already defectively nourished and with a diseased arterial system. A case is cited of a man who three years before had left hemiplegia with softening but with no involvement of the intellect. He suffered from an attack of facial erysipelas, which confined him to a hospital twelve days. At the end of this time he left the hospital cured of the erysipelas, but demented. The autopsy some days afterwards showed the circumscribed lesion, and also a diffuse lesion of the meninges and cortex.

2. *Stereotypy.*—It is important to study the motor manifestations of the insane. Among these some are due to disturbances of the motor apparatus, such as paralysis, contractions and convulsions; others are simply the reflex of a psychic state. It is these last, comprising for the most part fixed attitudes and repeated movements, that are united under

the name "stereotypies." The author follows with a historical résumé of the subject, and concludes that the different authors have comprised under the same denomination various conditions, so that the necessity for defining exactly what is to be meant by stereotypy becomes apparent. To do this we must distinguish fixed attitudes and repeated movements of catatonic origin on the one hand from fixed attitudes and repeated movements of demented origin on the other. These conditions, when due to catatonia, result in movements slow and uncertain, due to a state of stagnation and weakness. There is a defect of plasticitv in the movements, which are angular and mechanical. These manifestations tend to disappear with the acute stage of the disease. In the condition of dementia the cortical cells are wholly or partially destroyed. The resulting disturbance of motion is no longer functional, but the result of fragmentary associations due to the disappearance of association tracts and cells, and manifests itself by a tendency to motor uniformity. These phenomena are residual and secondary in origin, and have a basis in dementia. They are the ones the author has in view in this study of stereotypy.

Stereotypy, like tics, has its origin in conscious volition, but by continuous repetition the acts become automatic and persist long after the reason for their existence has ceased to be. This is a very similar process to the origin of normal automatic acts except that here the conscience never really has control of the acts as it does in stereotypy. The automatic movements of the pianist's fingers are still under voluntary control, can be made faster or slower, or cease altogether. The act of stereotypy, however, is lost to this control and recurs without reason.

The acts of stereotypy are acts which no longer, as formerly, have a reason, an aim. The mind has been by the processes of dementia, gradually disorganized, the various associations severed, the field of consciousness retracted, until finally the main image stands alone without the associations which were formerly a part of the system into which it entered. We see thus why the act, once started, should be repeated. Normally any act is the result of various tendencies, but there are no counteracting tendencies, because of the destruction of associations. For the same reason the act of stereotypy tends to express itself fatally. There is no delay in its execution. The battle of various tendencies which occurs in the normal brain, and is called "determination," "time of association," does not take place here; the act occurs with immediate and fatal precision. Stereotypy is motor representation deprived of all adaptation, representation which tends to fix itself indefinitely and to exteriorise itself fatally. WHITE.

(August, 1904.)

1. The Motor State of the Insane. VURPAS.
2. Note on a Case of Suicide. DAMAYE.

1. *Motor State of Insane.*—The importance of the motor state as an indication of the state of mind is well known. This article will attempt to study the disorders of the motor state, but will only include such as are psychical in origin and not those due to gross lesions of the lower centres, such as contractures, paralyses, choreic movements, etc. For the purposes of this study the motor act is divided into four stages. 1. Determination, or the condition antecedent to movement. 2. The mental representation of the movement or accomplished act. 3. Control, either permitting the production of the movement, or act or on the contrary, refraining from it and inhibiting it. 4. The execution.

An understanding of the results of the author's studies can best be obtained by reproducing the table at the end of this article, which enumerates the various disorders of motion observed in connection with these several stages:

A. Troubles of the condition antecedent to movement. Excess of intensity of the psycho-sensory processes.	Absence of dissimulation of the mental states in the expression and attitude. Motor expression of the object of delirium. Monologues and dialogues. Impulsions provoked by (*a*) delirium states. (*b*) hallucinations.		
B. Troubles of the motor representation. Too great intensity of the mental images.	(*a*) *Motor* exaltation. Exaggerated reactions.	Impulsions of degenerates. Tis and stereotypies of degenerates. Repetition of the same acts. Interruption by a movement of a certain pose. Arrest of an act at a certain movement. Repetition of the same movement during the execution of an act and before its completion. Reproductions of the same movements in certain determined conditions. Various troubles described under the denomination (délire du toucher). toucher). Catatonic attitudes of degenerates. Coprolalia, echolalia, stammering.	
	(*a*) Motor inhibition. Reactions diminished.	Motor arrest consecutive to certain obsessions. Aboulia of degenerates (by mental polarization). Abatement of the motor state of the obsessed.	
C. Troubles of the volitional consciousness. Diminution or absence.	Complete.	Motor imitation. Continuation of a movement commenced. Repetition of the same movement. Catatonia. Stereotypies of dements. Automatic movements of epileptics.	
		Impulsions.	of the epileptics, hysterics, traumatics, intoxicated, idiots, dements.
	Partial.	Motor troubles under their dependence.	Ideas of possession. Doubling of the personality. Psychomotor hallucinations. Medianism.
D. Troubles of motor execution.	(*a*) Too rapid.	Motor state in mania and maniacal states.	
	(*b*) Too slow.	Motor state in melancholia and melancholic states, of which the highest expression is:	
	(*c*) Arrest	Stupor. The aboulia of neurasthenics.	

2. *Suicide.*—The account of a patient who attempted suicide, and on examination showed absolutely nothing abnormal. Heredity was good; there was no obsession. She had attempted it while temporarily discouraged.

(September, 1904.)

1. Congress of Alienists and Neurologists.
2. Decimal Classification of Mental Diseases. TOULOUSE.

1. A report of the fourteenth congress of alienists and neurologists of France and the countries of the French language. The article is not of a nature to bear abstraction.

2. A scheme of the decimal classification of mental diseases following the general lines laid down by Deway and adopted in the international bibliographic institutes of Paris and Brussells. WHITE.

ARCHIVES DE NEUROLOGIE

(Vol. XVIII, 1904, No. 107, November.)

1. Epilepsy, Pathogenesis and Therapeutic Indications. PARIS.
2. Contribution to the Study of the Functions of the Optic Thalamus. BENAKY.
3. Two Cases of Merycism. RAVIART and CANDRON.

1. *Epilepsy, Pathogenesis and Therapeutic Indications.*—The first part of this article has been reviewed in a previous number of this journal. This review is concerned with the second half, viz., Therapeutic Indications in Epilepsy. The epileptic mother should not nurse her child, for if there is the least suspicion of epileptic taint, when such a mother nurses her child (and this is the rule, not the exception) not only is the child exposed to infantile convulsions, to epileptsic précoce, but the mother may easily suffer an aggravation of her own state. In any case we should aim to remove all causes which tend to produce states of abnormal excitement of the child's nervous system. Paris regards infantile convulsions as manifestations of true epilepsy in the majority of cases, as will be shown by investigation of the antecedents (epileptic or alcoholic history), a conclusion with which reviewer cannot agree. The institution of prenatal treatment (during intra-uterine life) will serve to prevent these convulsions, and assist the child to support the numerous and varied excitements which its neuropathic taint will surely bring about later. After weaning, a careful regimen must still be maintained, eggs and milk forming the diet staples. and gastro-intestinal excitations must be guarded against. Similar care should be exercised with the skin, kidneys and other emunctories; also the exhibition of vermifuges, if required. The first manifestations of irritability, ill-humor, etc., Paris regards as danger signals. Intellectual effort, though permissible, requires careful regulation, as does also physical exercise. The special irritability peculiar to the tainted nervous system of the congenitally predisposed requires the removal of all conditions capable of causing "nervous shock." (The fulfillment of Paris' program will necessitate a degree of vigilance and interest not available, unfortunately for the majority of cases.—D. I. W.)

Puberty accentuates the necessity for caution. Sexual irritability will require attention, cold baths, simple diet—if required, a mild course of potassium bromide. Erethism in both sexes is best controlled by camphor—often a mild hypnotic is in place. Warm baths, a little prolonged, are of service; as adolescence approaches, Paris advises that the cooperation of the epileptic (as is now done with the tubercular) be obtained, to the end that he may have an intelligent understanding of the consequences made possible by deviation from a vigorous hygienic life, both in the narrow sense as well as for society and his possible descendants in the broader. Paris thinks that the attention of the sufferer should be drawn to the habitual character of the epileptic, and in thus diverting his attention to the instability of the psychic manifestations, and to the accessions which he so often deplores, he may be taught how to avoid many sources of excitation. In thus opening the eyes of these patients to their true condition the force of the congenital taint (tare epileptique), as well as the effect of causes acting later, may be minimized. The alimentary regime in adult life, though naturally less restricted than during juvenile life, calls for similar control, and should be the more guarded the more the indications of excitement present. For the female a similar procedure will be required, but Paris emphasizes the importance of regulation of the menstrual function, inasmuch as there seems to be an especial relation between the menses and the occurrence of attacks. Both sedation and elimination are here called for. The sexual relations of the predisposed should be regulated both from the standpoint minimum and maximum natural indulgence, which control, of course, includes the proscription of all unnatural sexual life. The usual methods are mentioned. Paris has discussed the prophylactic side in an extended way, because, as he says, so much attention has been paid to the treatment of the attack and so little to the intervals, or the disease as a whole.

Curative Treatment.—Paris establishes four principal indications: (1) Diminution of the meningo-encephalic impressionability; (2) moderation of the functional activity of the thyroid and genital apparatus; (3) regular elimination of their secretions; (4) removal of all complementary causes of excitement (meningo-encephalic); as the development of toxines. In infancy the curative treatment has less extended indications. Modification of thyroid activity would involve possible danger to psychic and physical development. On the other hand, the causes of excitement are less in evidence, especially if a proper regime has been adopted. To diminish cortical excitation the author still relies chiefly on the bromides, but often gives in connection with them trional in two doses. half at 8:30 to 9 P.M., half next morning. He speaks highly of trional, and in cases of bromide saturation recommends it as a temporary substitute.

As for the second condition—moderation of functional thyroid and genital activity, Paris thinks that excess, or lowering of thyroidal function, has analogous accompaniment in the genital sphere. Camphor, lupulin, warm baths (slightly prolonged), diuretics, and diaphoretics are mentioned. Paris prefers camphor in conjunction with a hypnotic, in females, before the menstrual epoch. The epileptic is often an "arthritique," or his various conditions strongly resemble those of the arthritic type. In searching for a remedy which can prevent the accumulation of glandular secretions peculiarly noxious when acting on the arthritic type of epileptic, Paris has fixed his choice on benzoate of lithia. This brings about, in Paris' opinion, a sort of lavage of the blood, preventing the accumulation of thyroid or other glandular secretions therein. Regulation of all the functions concerned in excretion is, of course, imperative. Intestinal antiseptics have a place. Camphor is recommended as combining the latter quality with its well-known anaphrodisiac action. Serum therapy does not meet with the author's unqualified approval.

2. *Functions of the Optic Thalamus.*—Report of a case in which there were constant movements of a menacing nature, accompanied by a spoken threat of striking. Accompanying these was a forced mocking smile, the effect of which was accentuated by a ptosis (right eye) and external squint. Other symptoms present in addition to the spasmodic movements in the right arm. besides those above noted, were paralysis of the lower extremities, inability to stand erect, augmented reflexes, intellectual torpor.

Autopsy revealed a glio-sarcoma of the parietal lobe which invaded the frontal lobe and had involved, by a downward prolongation, the posterior part of the optic thalamus, the internal capsule, nucleus lenticularis and external capsule. Attention is especially directed to the spasmodic menacing movement by the appropriate word and the forced, almost mocking, smile, phenomena which belong to the domain of mimicry. Benaky reviews the case of Nothnagel wherein with preservation of voluntary contraction of the facial muscles there was inability to laugh. In this case, as in Gowers' case, there was destruction of the thalamus. On the other hand, Yimoucopoulo reported a case where, along with the facial paresis, there were spasmodic movements and continual forced laughter. In this case the autopsy showed a small cyst in the posterior portion of the thalamus. He thinks irritative lesions in this region may cause forced movements of the facial mimic muscles; destructive lesions, on the other hand, paralysis of the mimic movements of these muscles. This pathological gaiety was explained by Oppenheim and Eisenlohr as a consequence of an irritative lesion of the thalamus. Benaky presents his case as one due to irritative lesion.

3. *Two Cases of Merycism.*—Raviart and Caudron report two cases of merycism, the details of which are quite interesting, but no conclusions are drawn.

WOLFSTEIN (Cincinnati).

JOURNAL OF MENTAL SCIENCE

(Vol. L., 1904, No. 211, October.)

1. Presidential Address on Paranoia. Sixty-third Annual Meeting Medico-Psychological Association. R. PERCY SMITH.
2. The Psychology of Hallucination. W. H. B. STODDART.
3. Histological Studies on the Localization of Cerebral Functions. ALFRED W. CAMPBELL.
4. Educational Treatment of Young Epileptics. G. E. SHUTTLEWORTH.
5. Statistical Tables. CHAS. A. MERCIER.

1. *Presidential Address on Paranoia.*—In this address, which is a most able review of the whole subject, Smith traces the development of paranoia and contrasts its position in the psychiatric classifications of the various nations. He particularly emphasizes the conservative position of the English school on this subject, and shows most interestingly that even in the country of its origin there is no common agreement as to the connotation of paranoia. Smith's conclusions are as follows:

(1) The term "paranoia" is useful if it be limited to cases of chronic delusional insanity in which there are organized and systematized delusions, whether of persecution or exaltation, and whether these run separately, concurrently or by transformation from persecution to exaltation, and whether the disorder originates in childhood and youth (originäre paranoi), or later i nlife (tardive paranoia), and whether associated with heredity or not.

(2) In all these cases the importance of the affective element of mind must be ignored, and it is erroneous to use the term "paranoia" as implying primary intellectual disorder to the exclusion of, or prior to, disorder of "Affect."

(3) Allowing that there are acute cases in which delusions appear to be organized and systematized, and yet in which recovery seems to take place many of these cases are merely the initial phase of chronic delusional insanity with a remission of symptoms.

(4) If the incubus of the idea of primary intellectual disorder be got rid of, there is no difficulty in recognizing that some cases of paranoia may begin with an acute functional mental disorder of the nature of

melancholia or mania (as is indeed recognized even by those who take the primary intellectual view), or even may follow a delirious or confusional state.

(5) With this exception, acute confusional insanity (acute Verwirrheit) and acute delirious states (acute delirium, collapse-delirium, Erschöpfungs-delirium) should be regarded etiologically and clinically, and from the point of view of diagnosis and prognosis, as entirely apart from paranoid or chronic delusional insanity.

(6) Mercier's term "fixed delusion" should be used for states secondary to acute forms of insanity, where the persisting delusions are not organized or progressively systematized.

(7) With regard to terminal dementia in paranoia, it is trying to prove too much to say, as some authors do, that dementia does not ever supervene in this condition; and I think that Kraepelin's action in removing a large group of cases in which terminal weak-mindedness occurs from the domain of paranoia to that of dementia præcox is open to question. There seems to me a possibility that dementia præcox, with its hebephrenic, catatonic and paranoid forms, may become the new universal disease (Universalkrankheit), into which a large number of cases may be thrown, and which will give rise at no distant date to as much discussion as has attended Paranoia.

2. *The Psychology of Hallucination.*—The author calls attention to the points of dissimilarity between percepts, ideas, illusions, and hallucinations. Their points of resemblance are psychological, their points of difference are mainly physiological. Perception and ideation localize an object and give it a shape occupying a certain amount of space. Percepts and ideas are in reality only abstractions. The study of perception is, therefore, in reality but little more than the study of perception of space. In the visual domain, *e. g.*, the most obvious difference between perception, ideation, illusion and hallucination, is that while in perception and illusion there is a stimulus to the peripheral sense-organ (retina), in ideation and hallucination there is no such stimulus. In perception and illusion the stimulus to the angular gyrus arrives by way of the optic radiations, occipital lobe, and occipito-angular association fibres; in ideation and hallucination the stimulus reaches it by way of other association fibres than the occipito-angular bundle. This is evidenced by the existence of visual hallucination in the blind, auditory hallucination in the deaf, etc. In the visual hallucination there is a negative as well as a positive side to the process. The positive side is the seeing of the hallucination image; the negative side, failure to see neighboring objects. The interpretation of the negative part is that the neurones which normally conduct sensations from the retina are dissociated from each other. The positive side of the hallucination process is due to stimuli reaching the centre by way of association fibres other than those by which sensations are transmitted from the peripheral sense organ. Absence of sensations of other modalities than that affected favors the hallucinated state. The absence of other stimuli allows the affected sensory area to dominate consciousness. Hallucination depends, then, upon two factors, diminution of sensation and disturbance of association, which factors are of variable prominence.

Head has recorded cases in which hallucination was associated with the pain of visceral disease. Presuming that there was no anesthesia in these cases, Stoddart suggests that the continued painful sensations spread by way of association fibres to distant sensory areas of the cortex, giving rise to visual, auditory, and other hallucination. Charts are given showing marked analgesic areas of the disturbance (and, therefore, presumably anesthetic) in the insane. Stoddart suggests that epigastric and other allied sensations arise in those cases in which, owing to some affection of the cerebral cortex, there is some loss of sensation. When, therefore, any considerable part of the body is anesthetic, consciousness is dependent on the sensitive remainder. The sensitive remainder dominates consciousness, has "greatness thrust upon it." In the epigastric aura of epilepsy Stoddart reasons as follows: Loss of consciousness is the first symptom. It is said all sensation is lost because of the unconsciousness, but Stoddart thinks the patient becomes unconscious because he has lost all sensation. He suggests that the sensory defect begins at the periphery (arms and legs), and allows the epigastric sensitive remainder to dominate consciousness; hence the aura. The last event as the patient falls is the loss of epigastric sensation. The neural interpretation is the dissociation of neurones between the peripheral sense-organ and the cortical sensory centre. In conclusion Stoddart compares perception, ideation, illusion, and hallucination with regard to the manner in which in each process the cortical centre is stimulated. In perception, stimulus is transmitted from the periphery; there is neither *trans-cortical* association nor peripheral dissociation. In ideation the centre is stimulated by way of association fibres, but again there is no dissociation from the periphery. In illusion the centre is stimulated on the one hand by way of association fibres, and on the other from the periphery. The physiological difference between ideation and illusion is this, that the peripheral stimulus *does not interfere with* ideation, but is *necessary* to illusion. In hallucination there is trans-cortical association and peripheral dissociation. It differs essentially from perception, ideation, and illusion, in that the essential factors of hallucination is its negative factor.

3. *Localization of Cerebral Functions* (*Alfred W. Campbell*).—Campbell has made extended histological investigation into the structure of the cerebral cortex. He utilized normal and also pathological material. He also compared the cortex of two members of the anthropoid ape family and several of the lower animals. The brain harbors two varieties of centres: "primary," common to all animals and essential to survival, viz., centres for movement and common and special sensation; and "higher evolutionary," concerned in the higher psychic functions which distinguish man. The "motor" area is as easy to recognize histologically as experimentally. It is confined to the pre-central and does not spread to the post-central gyrus. It is mainly characterized by the giant cells of Betz. Proof of the pathological side is afforded by the findings in amyotrophic lateral sclerosis, and in the brains of individuals who have undergone amputations. The sulcus cruciatus of the lower orders is not the homologue of the Rolandic fissure. An isolated fissure on the posterior limb of the sigmoid gyrus forms a sharp histological boundary between the motor and what Campbell regards as a common sensory area. This he regards as the homologue of the Rolandic fissure, which is not a dividing line separating the motor area into halves, but the posterior boundary of the motor area. Motor representations are in three levels (Hughlings Jackson). Simple movements in the anterior cornual spinal cells; secondly, primary automatic movements of more complex type in the giant cells of the pre-central gyrus. Campbell places the third centre for skilled or higher evolutionary movements in a zone immediately adjoining the pre-central, called by him the "intermediate pre-central." It embraces two important centres for skilled movement: at its foot Broca's speech area, and above this the cheiro-kinesthetic centre for writing. This strip also contains a series of centres for skilled movements connected by commisural fibres, with the automatic simple centres in the pre-central gyrus. The rest of the frontal lobe is divisible into two areas, which are termed "frontal" and "pre-frontal." The pre-frontal subdivision is structurally extremely weak, and that it is so functionally is further proven by its electric inexcitability and by clinical experience. Histology supports the view that phylogenetically it is the very last pal-

lium to appear. The cortex, which is comprised under the "frontal" subdivision, is more extensive in man than in the ape. Campbell does not dispute Flechsig's doctrine that the frontal lobe must be a higher association centre, but accepts cautiously the evident support afforded by the atrophy in this region in cases of dementia, inasmuch as the cortex of the frontal lobe is built upon an extremely weak and collapsible framework of nerve fibres, especially when compared with the central gyri, occipital lobe and other parts. Cortex of the parietal lobe. Campbell "plumps" for the view that the post-central gyrus is the main terminus for common sensory impressions. Histologically the difference between the two gyri which are divided by the Rolandic fissure is even microscopically noticeable. Further, he advances in support of the sensory nature of the hinder gyrus, first, early medullation, as in the posterior tracts of the cord; second, the work on secondary degeneration of the cortical lemniscus (upper sensory neurone); and third, changes which he finds in the post-central gyrus in tabes, a disease as clearly restricted to the sensory neurones as amytrophic lateral sclerosis is to the motor.

Adjoining the "post-central" area proper, which may be regarded as a primary centre for the recognition of the simplest components of common sensation, is an area containing higher evolutionary "sensory centres," in which lesions may give rise to isolated disturbances of higher sensory components, *e. g.*, astereognosis, loss of muscle sense, etc., sensory components involving a higher psychic process. These are included in the "intermediate post-central area." Between this "sensory" area in front and the "visual" area is a vast area embracing Flechsig's posterior association area, an area in close anatomic relation with all forms of sensory components, both common and special.

This area is probably a field for the further elaboration and interpretation of impressions primarily received by the various sensory areas. In phylogenetic development the progressive increase in size of the frontal lobe, which Hitzig contended was proportionate to intellectual capacity, *is not more marked than a similar progressive increase in the expansion of the parietal lobe.* In upward evolution both frontal and parietal lobes undergo equal expansion. An interesting point made by Campbell is that in agenesis the parts most prone to suffer are those which are phylogenetically youngest. In three cases cited (idiots) all the motor and sensory centres were well developed, but the frontal gyri anterior to the pre-central and the parietal behind the post-central areas showed marked microgyria. The visual cortex is divisible into two areas; one follows closely the calcarine fissure (marked by the line of Gennari), and is probably the primary centre for visual impressions; the second, an investing area called the visuo-psychic. serves for further elaboration of these impressions. The primary auditory area Campbell locates in the transverse temporal gyri, while the audito-psychic centre corresponds in distribution to the well-known "word-hearing" centre. Two plates embodying these views accompany the paper.

4. Do not require reviewing.
5. Largely critical; not adapted for review.

WOLFSTEIN.

MISCELLANY

OPTIC NEURITIS AND FACIAL PARALYSIS. E. A. Shumway (Journal A. M. A., Feb. 11).

The author reports a case of post-papillitic optic atrophy with a history of prior right-sided facial paralysis with pain in jaw, and with a noticeable flattening of the right side of the face from loss of subcutaneous fat, together with enophthalmus, all on the right side, while the optic atrophy was bilateral, most marked on the left. He finds in literature only seven similar cases of this association of facial paralysis and optic neuritis, though a number of cases of optic neuritis have been reported in connection with polyneuritis. The atrophy and sinking of the eyeball is evidently rarer, as he has found no reports of a similar case. He has, however, been able to examine a case of Dr. Spiller's with flattening of the face and enophthalmus following rheumatic facial paralysis, and implying, he thinks, as in his own case, some involvement of the seventh nerve. There were chloroanemic and disordered menstrual symptoms in Shumway's case, but he does not attribute to them the optic atrophy. His conclusions are given as follows: "1. Optic neuritis is occasionally associated with facial paralysis, either alone or as part of a multiple neuritis; the etiologic factor may be rheumatism, but at times appears to be infection, the nature of which is as yet undetermined. The optic neuritis is usually of the retrobulbar type, but a decided papillitis may be present, and be followed by more or less marked atrophy. In cases of multiple neuritis of the cranial nerves the eye grounds should be examined for possible optic nerve complication. 2. In facial paralysis, flattening of the face and enopthalmus may appear, and are to be considered as due to a neuritis of the fifth nerve, and not to involvement of possible sensory fibres in the facial nerve."

BLANK CARTRIDGE TETANUS. D. H. Dolley (Journal of the A. M. A., Feb. 11, 1905).

Dolley has investigated blank cartridges from several makers with special reference to their bacteriologic contents, employing cultural and incubation, as well as microscopic methods. The findings were rather negative as regards the tetanus bacillus, but the *Bacillus aerogenes capsulatus* (Welch) was present in a large proportion of the cartridges examined. Notwithstanding this fact, tetanic symptoms developed in a number of animals inoculated, and in still other animals inoculated from cultures from these. His conclusions are: 1. *B aerogenes capsulatus* (Welch) is present in a large proportion of the wads of the three makes of the cartridges examined. 2. The wads of the Peters Company, inoculated in rats, guinea-pigs and rabbits, produced characteristic symptoms of tetanus. 3. The powder of the three varieties of cartridges examined was negative for *B. tetani* and *B. aerogenes capsulatus*. 4. His efforts at isolation of *B. tetani* from the wads have so far been unsuccessful. 5. There is abundant evidence, from clinical observations and animal experiments, that the wads of certain blank cartridges contain *B. tetani*. He says that Dr. Welch told him that he considered it diagnostic to see an animal in convulsions.

CHOREA. W. G. Spiller (Journal A. M. A., Feb. 11, 1905).

Spiller thinks that the relation of chorea to rheumatism has been greatly over-estimated. In most of his cases he could not detect it. He also has not been able to recognize any peculiar facies of the disease; nor does he agree with Gordon and Eshner that there is any peculiar characteristic of the patella reflex in chorea. The arsenical treatment of the disease does not seem to be without disadvantages, and should be watched very closely. He has seen arsenical neuritis and idiosyncrasy. The pathology of the disorder is still obscure. The "chorea bodies" are not characteristic. Apoplectic hemihypertonia is distinct from athetosis; the spasm is tonic, unilateral, associated with a little weakness, but not with contractures; develops after an apoplectic attack, and is probably due to irritation of the motor fibres below the cortex. Spiller does not accept Kahler and Pick's theory of the choreiform movements being caused by irritation of the pyramidal tract. It is hard to understand the comparative rarity of hemichorea if this were the case.

Convulsive Tic. H. T. Patrick (Journal A. M. A., Feb. 11, 1905).

Convulsive tic may be said to be a habit spasm, a sort of motor expression of an imperative impulse. It may develop from some peculiar motion incident to the patient's occupation, but its original cause is generally sensory—some uncomfortable sensation which an attempt is made to relieve by a movement which finally becomes habitual. It does not affect voluntary movements, is diminished by quiet, rest or mental diversion, and is aggravated by self-consciousness, observation, excitement, etc. The prognosis varies. In children it is ordinarily good, but in adults it is often rebellious. The patients are generally nervous and unstable, and in cases of children, unwise parents and rearing are often responsible. With them the habit may be broken by judicious diversion or correction. With adults the treatment is apt to be unsatisfactory, but Patrick thinks the soporific treatment, keeping the patient asleep for two or three weeks at a time, using hypnotics judiciously with frequent changes of the drug, followed by the educational exercises of Brissaud, will be found to be most effective in the spasmodic torticollis of the adult.

Hysterical Movements. H. T. Pershing, Denver (Journal A. M. A., Feb. 11, 1905).

The author gives the diagnostic points of hysterical movements as compared with chorea and convulsive tic. One characteristic is that they are always movements which can be produced voluntarily, though this also may be the case with convulsions from organic disease. The more regular the movement the greater the probability that it is hysterical, but the possibility of hysteria complicating other conditions must not be forgotten. The most characteristic movement it a rhythmic oscillation involving one part, and next are certain highly coördinated movements, such as jumping or dancing, with or without impairment of consciousness. Chorea may simulate hysteria and be due to similar emotional causes, and the diagnosis may be difficult. Hysterical movements are more likely to be regular and grouped in distinct paroxysms and to have more of the staccato movement, but most of the rules for distinguishing these diseases require qualification. Hysterical movements of a limb may simulate Jacksonian epilepsy, but there is no rise of temperature, no paralysis nor mental deterioration. Prognosis and treatment must be guided by general principles. A cure is always possible, though the condition may be obstinate. Moral treatment is imperative. If the patient's mental processes cannot be happily directed, everything else will be useless. If they are so directed, the rest will be easy.

Chorea in Pregnancy. J. M. Semple (Northwest Medicine, Dec. 1904).

This type of chorea uncommon. Occurs about once in a thousand pregnancies. Prognosis unfavorable, as compared with Sydenham's chorea in children, the death rate varying from 17 per cent. to 36 per cent. with the former type, while death rarely occurs with the latter. Disease, in majority of cases, occurs for the first time in primiparæ. Symptoms may occur any time from conception to ninth month, but more frequently found during the first three. Unless as a secondary attack, the later the onset the graver the prognosis. In chorea of pregnancy. interference in speech, deglutition, and sleep, more marked than chorea of childhood. Mental disturbance often of maniacal type.

J. E. Clark (New York).

Clinical Studies in Arterio-sclerosis. Alfred Stengel ((The Cleveland Medical Journal, December, 1904).

Wide divergence in the nature of arterio-sclerosis developed in presenile period, as a result of more or less well-recognized causes, and that which occurs during true senile involution. Senile form of disease is one of a process that is essentially diffuse and, perhaps, involves the extra vascular tissues simultaneously with the vascular apparatus, while the pre-senile form, or pathologic arterio-sclerosis, is, in the beginning, a disease of the blood-vessels alone. Rapid degenerative changes in the organs, possibly due to restricted involvement of vessels and the character of lesions, are more characteristic of the pre-senile than the senile type. Later stages of senile form, and to less extent with pre-senile type, patients' color would suggest cachexia or high-grade anemia. Blood examination in these cases shows no commensurate reduction in number of corpuscles or of the coloring matter. Pallid appearance, thought to be due to the contracted vascular channels. Marked fluctuation in quantity and variability of specific gravity of urine quite noticeable. Paroxysmal sweating a symptom in many cases during earlier stages of disease. Author's observations as to blood pressure have shown comparatively high diastolic pressure, thus during the earlier period there is a peculiar prolongation of the first heart sound. J. E. CLARK (New York).

MENTAL SYMPTOMS ASSOCIATED WITH PERNICIOUS ANEMIA. William Pickett (The American Journal of the Medical Sciences, June, 1904).

From an examination of seven cases of paresis the author draws the conclusion that when the spinal cord bears the brunt of the disease process a simple anemia with leucocytosis is found; when mental symptoms alone appear the blood state is not characteristic, and from this it appears that perhaps anemias, or the toxins of anemias, are a factor in the cord lesions even of paresis. Abstracts of five cases of pernicious anemia are given, and a composite picture of the mental disturbance in these cases presents a shallow confusion with impairment of the ideas of time and place, more marked on awakening from sleep. The patient fabricates, relating experiences of "yesterday" in a circumstantial manner. Illusions, particularly of identity, are common. Hallucinations of any of the senses appear at times. Persecutory delusions may arise. They are usually transient, causing episodes of fear and agitation, but they may persist for considerable periods. Korsakoff's disease and *folie Brightique* resemble it closely. C. D. CAMP (Philadelphia).

TABES DORSALIS AND ITS RELATION TO SYPHILIS. Lesser (Berl. klin. Woch.., Jan. 25, 1904).

The author discusses the connection between locomotor ataxia and syphilis, chiefly from the pathological side of the question, and attempts to bring further proof from the post-mortem room that tabes is a syphilitic affection. In the first place, in answer to the question, How many persons who have had tabes show post-mortem signs of undoubted syphilis, and how many persons who have died over 35 years of age show signs of undoubted syphilis post-mortem? he found that 28 per cent. of the tabes cases had post-mortem signs, while of all persons who died over the age of 35 only 9.5 per cent. showed svphilitic signs. He realizes that a number of cases of former syphilis will not leave any signs behind, but it is clear that tabes and syphilis stand in a causal relationship together, at all events in some cases. The next question which he sets himself to answer is: Is the tabes to be regarded as a direct syphilitic affection in those cases in which the connection can be ascertained? In order to reply to this question, he finds it necessary to go fully into the pathology of syphilitic lesions. The changes may be best divided into: (1) Simple hyperplastic growths, having the characters of inflammation, and which leave no anatomical changes behind; and (2) gummatous growths. One must recognize infiltrating papular inflammations and proliferating interstitial inflammations. He therefore classifies syphilitic lesions into the following: (1) Papular inflammations, being equivalent to secondary syphilitic lesions and affecting the skin chiefly; (2) gummatous inflammations, being equivalent to tertiary manifestations, and may affect any part of the body; and (3) interstitial inflammations, which he calls quartary manifestations, and which only affect the internal organs. The first class reacts to mercury, the second to iodide of potassium, and the third is refractory towards both. As examples of interstitial changes, he quotes fibrous orchitis and syphilitic fibrosis of the liver. While the secondary changes end in absorption, the quartary changes are irreparable. After discussing the histological changes in these manifestations, he passes on to consider the changes found in tabes, and divides these into, first, parenchymatous degeneration of the nervous elements of the posterior columns of the spinal cords; and secondly, overgrowth of the interstitial tissue of the cord, the so-called neuroglia. The rest of the histological changes are not constant, and are, therefore, of minor importance. In deciding which of the two are primary, he argues by analogy that the interstitial changes are primary. He points out that in cirrhosis of the liver the cell degeneration is always regarded as secondary; so in chronic interstitial nephritis and in atrophia lævis of the tongue. He therefore argues that tabes is a quartary manifestation of syphilis. He next meets each of the arguments raised against the suggestion that tabes is a syphilitic affection. That it does not react to mercury or iodide of potassium is explained, since none of the quartary manifestations do. The second objection is that in the majority of tabes cases there are no traces of other co-existing syphilitic lesions of the skin or internal organs, and conversely, in the severer cases of syphilis tabes dorsalis very rarely is found. In reply to this, he regards the manifestations of syphilis as being due to a micro-organism, the virulence of which appears to diminish in the course of time. While the virulence is great, the lesions caused are of the nature of secondary manifestations, and only when this has become much attenuated are quartary changes produced. Thus it is not likely when interstitial changes are found that gummatous or simple inflammatory changes will also be present. The third objection is that the extraordinary frequency of syphilis, when compared with the rarity of tabes speaks against a connection between the two diseases. He points out that all the late manifestations of syphilis are rare, and if one compares the number of syphilitics who suffer from a gummatous affection of the elbow-joint one will find that the percentage is still less than in the case of tabes. The fourth objection is that sufferers from tabes can infect themselves with syphilis. That a want of immunity cannot be taken as proof against the previous existence of syphilis is shown by the fact that syphilitics with tertiary manifestations at times have acquired fresh syphilis. The last objection is that tabes is a system disease, and that these diseases cannot be syphilitic. According to Lesser, the characteristic of interstitial syphilitic changes is that they have a predilection for certain localizations, and that nowhere is this more clearly shown than in syphilitic fibrosis of the liver. In conclusion, he remarks on the coincidence of tabes and aneurysm. He finds that every fifth patient suffering from tabes dorsalis shows signs of aneurysm. From its histological characters, it appears that the majority of aneurysms are interstitial syphilitic manifestations, and this he considers is evidence in favor of his views. JELLIFFE.

EXOPHTHALMIC GOITER SUCCESSFULLY TREATED WITH ANTITHYROIDIN.

Möebius's favorable results from the employment of antithyroid serum in exophthalmic goiter induced Dr. Josionek, of Mildenau-Wiesenbad, to try the serum in two typical cases of the disease. Both patients showed the characteristic symptomatic trinity: exophthalmos, tachycardia, and increase in the size of the thyroid; there was also a distinct tremor, general restlessness, and insomnia. One patient, a man of fifty-four years, had noticed the first symptoms two months prior to treatment. For the first week the patient was treated hydrotherapeutically—cold tubing over neck and heart and compresses around the body. This treatment improved the insomnia, the pulse was reduced from 112 to 93, but the size of the neck, which measured 43 centimeters, remained uninfluenced. Then antithyroidin-Möbius, 1 gm. (15 grn.) twice a day, later on 2 gm. (30 grn.) twice a day, in a little milk, was given to the patient. From April 25 to May 8 patient took in all 30 gm. (1 oz.) of the antithyroidin. The thyroid became distinctly softer, the size of the neck diminished by 6 centimeters (2½ inches), the tremor had almost disappeared, there was hardly anything abnormal in the position of the eyeballs, the pulse rate was reduced to 78 per minute, sleep was good, and the general condition and spirits of patient were excellent.

The second patient, a slender woman of fifty-one, had noticed a swelling in her neck towards the end of her school years. As a result of severe excitement on account of financial loss in 1903, the swelling increased; at the same time the eyeballs began to protrude and heart trouble set in. The patient used various remedies under medical advice, and among other forms of treatment she drank milk from thyroidectomized goats for ten weeks, but her condition remained uninfluenced. When she came under the author's treatment on June 5 she presented the following symptoms: Pronounced exophthalmos, restlessness, pressure in the head, tremor; circumference of neck, 35 centimeters; pulse, 96 per minute. The patient took 2 gm. (30 min.) of antithyroidin a day for ten days, with no notable improvement; after an interval of about eight days the serum was again given to her in double the dose, namely, 2 gm. (30 min.) twice a day (1 dram daily). As a taste corrigent she used strawberry lemonade. In the course of six days there was a distinct improvement in the general condition, as also in the special symptoms of the disease. After the use of another 30 gm.—50 gm. in all—the very hard thyroid became quite soft, the circumference of the neck diminished 2 centimeters (4-5 inch), and the pulse-rate was reduced to 82 per minute. There was only a slight change in the position of the eyeballs. The patient was satisfied with the result, having improved bodily and mentally to such a degree that she was enabled to undertake a voyage.

These two cases prove the favorable influence of antithyroidin-Möbius on Basedow's disease, and they also show that recent cases are more readily influenced than old ones. The antithyroidin contains one-half of 1 per cent. of phenol, for the purpose of preservation, but the author has not noticed any injurious effect from this addition, thus corroborating Möbius's statements in this respect. The antithyroidin is prepared by E. Merck, of Darmstadt, from thyroidectomized sheep, and is obtainable in glass containers, 10 gm. (2½ drams) each.—*Med. Woche*, 1904, No. 37.

Jelliffe.

Book Reviews

JAHRESBERICHT UEBER DIE LEISTUNGEN UND FORTSCHRITT AUF DEM GEBIETE DER NEUROLOGIE UND PSYCHIATRIE, VII., Jahrgang. Bericht über das Jahr., 1903, Zweite Halfte. S. Karger, Berlin.

We have had occasion in the past year to notice the first half of this year book, and to comment on the wisdom of the publishers in expediting its appearance by the method of subdivision. Year books are usually so far behind at best that any method that aids in overcoming a part of this difficulty at least is welcome.

Apart from the advantage outlined, we find the volume fully as useful as its predecessor. It is invaluable to the workers in neurology and psychiatry, not alone because of the great saving in time that its use ensures, but it is also a great educator, presenting as it does in compact and readable form the views of workers in all parts of the world.

The sections on psychiatry are greatly enlarged, reflecting the wider interest taken in this branch of medicine, and new sections on Criminal Anthropology and allied branches have been added, or expanded to meet the broadening of the study of "mankind." Altogether, the work is a great credit to its editors, collaborators and publishers, and we commend it for its practical and thorough summary of the main facts bearing on neurology and psychiatry which were brought to light during the year 1903.

JELLIFFE.

ARBEITEN AUS DEM NEUROLOGISCHEN INSTITUTE AN DER WIENER UNIVERSITAT. Herausgegeben von Prof. DR. HEINRICH OBERSTEINER. XI. Band. Franz Deuticke, Leipzig und Wien. 25 Marks.

Professor Obersteiner maintains his activity in this the eleventh volume of his Arbeiten, and there are many valuable studies from his pupils that bear witness to their industry and interest in neurological science.

The present volume of 442 pages contains fifteen separate studies. Of these six are strictly anatomical. One of these is a study of comparative anatomy, that of E. Popper, who deals with the Spinal Cord in Marsupials. Six are pathological, in the narrow sense, whereas the remainder may be said to be clinical.

Of the more distinctly clinical studies that of Neurath, on the Nervous Complications of Whooping Cough, is particularly valuable. This disease has been neglected by the neurologist for some time, and Neurath's complete study is opportune and suggestive. Chronic Idiopathic Hydrocephalus is written on by A. Fuchs, and Professor Obersteiner contributes a short and interesting study on Fatty Pigmentation in the central nervous system. Porencephaly is by Messing. The Parasitology of the Brain, by Bunzi, is a very suggestive and complete paper. It promises to be one of lasting value on this little developed subject.

We cannot mention other papers in this series, but they are all well worth the time and trouble put upon them. GOODALE.

THE LAW AND THE DOCTOR. VOL. II. THE PHYSICIAN AS WITNESS. Arlington Chemical Co., Yonkers, N. Y.

Almost every member of the medical profession is sooner or later called upon to appear as a witness, either in the capacity of an expert or otherwise. This pamphlet gives in clear and complete form the information which will assist a physician so called upon to discharge his duty in the most efficient manner, since it points out matters which might not occur to one inexperienced in such legal processes, but of the utmost importance to enable him to have a definite idea of his own rights and obligations. Such matters as "Attendance at Court: How Compelled," "Admissibility of Evidence," "Competency and Relevancy of Testimony," "Examination and Cross-Examination," etc., are treated in turn in a helpful and thorough manner.

JELLIFFE.

MULTIPLE PERSONALITY. An experimental Investigation into the Nature of Human Individuality. By BORIS SIDIS, M.A., Ph.D. (Harvard). Author of the Psychology of Suggestion and Psychopathological Researches, and SIMON P. GOODHART, Ph.B., M.D. (Yale). D. Appleton & Company,

If one case, as in this work, furnishes sufficient data and inspiration for a book of 456 reading pages, how is clinical medicine of the future to be provided library room? One shudders here at the application of the rule of three.

. Part I. of this book is by Dr. Sidis, and deals with an interesting anatomic and physiologic study of personality, although the treatment is somewhat elementary. The fear that some of us entertain lest the neurone theory be discarded, does not seem to disturb the writer, who builds a normal and morbid physiology of personality upon it.

Part II., by both authors, is chiefly interesting because of the careful record of a case of double personality—the Hanna case. No effort has been made to spoil the dramatic quality of the case, as shown by the chapter titles—"Revelations from Dream Life," "Upheavals of the Subconscious," "Resurrection of Outlived Personalities," "The Struggle and Union of Alternating Personalities." The possibilities of scientific study in this case of traumatic amnesia and its termination in recovery, are sufficiently stimulating to the neurologist even without the literary and dramatic charm of the case setting. One is tempted, however, to speculate on the relative rapidity with which the case would naturally have recovered without the annoyance of the psychic irritation practised.

Part III., containing facts and fancies relative to consciousness and mental possibilities, quite outdoes Part I. in psychic dramatization. Thus some of the chapter headings are: "The Fading Moments," "The Brightened Moments," "Transmutations of Subconscious Messages," "The Process of Mental Resurrection," "The Reawakening," "Hypnoidic States or Resurrected Lives," "Hypnoid States or Underground Life," "The Twilight of Consciousness or Dämmerzustände."

The work on the whole shows an earnest attempt of the authors to present the importance of prolonging the transition state in alternating personality cases, to the end that a fusion of the multiple into a single and natural personality may finally result. However serious one may strive to regard the authors' methods and success in the Hanna case, one must be guarded as to their general application to all cases, and be especially chary of a summer's advent, which is heralded by a single swallow. This warning is particularly true in so-called applied psychology, which may still be regarded as not without the realm of psychic chance.

L. P. CLARK (New York.)

A DICTIONARY OF NEW MEDICAL TERMS. By GEORGE M. GOULD, M.D. P. Blakiston's Son & Co., Philadelphia.

This volume is in the nature of a supplement to the author's "Illustrated Dictionary of Medicine, Biology and Allied Sciences," published ten years ago. The special necessity of such a supplement is evidenced by the fact that more than 30,000 new words have been devised in the past decade for employment in the sciences named. Dr. Gould has a lively realization of the difficulties that beset the lexicographer in general and the medical lexicographer in particular, and very definite ideas of the goal he should strive to reach. He brings to his task a broad and tolerant scholarship which recognizes the value, historical and otherwise, of even words which might better have been left uncoined. He understands that a dictionary is to a large extent a record of what is, rather than of what ought to be, and while he by no means "lets down the bars" for a horde of unauthorized expressions, he does aim to make intelligible any likely to be encountered in medical work. He unmistakably possesses that "feeling for words" which is a prerequisite to successful handling of them in any field. The book is appropriately bound in a substantial and dignified style.

JELLIFFE.

A THESAURUS OF MEDICAL WORDS AND PHRASES. By WILFRED M. BARTON, M.D., Assistant Professor of Therapeutics and Materia Medica, and Lecturer on Pharmacy, Georgetown University, and WALTER A. WELLS, M.D., Demonstrator of Laryngology, Georgetown University; Adjunct Professor of Laryngology, Washington Post-graduate School, etc. W. B. Saunders & Co., Philadelphia, New York and London.

This thesaurus deserves its name, for it is a veritable treasury for those—and they are many—who do not possess the natural gift for words, the facility in their use and recollection which is an indispensable requisite to fluent and effective speech or writing. Whether the need is for synonyms to vary the author's diction, or for a form of expression for a vague and nebulous idea, or for a dignified and technical substitute for a popular phrase, this volume will supply it. The arrangement is admirable, and the choice of type such as will greatly facilitate the use of the book.

GOODALE.

THE DOCTOR'S RECREATION SERIES. Edited by CHARLES WELLS MOULTON. Volume III, "In the Year 1800." By SAMUEL WALTER KELLEY, M.D. The Saalfield Publishing Co., Chicago, Akron, Ohio, and New York.

The third volume of this series appears in the same attractive dress as its predecessors ,and presents in the form of a novel the experiences and adventures of one "Dr. Brush," who is alleged to have lived and flourished, as the title indicates, about the year 1800. The author is himself a physician, and undoubtedly the first, although not the last, to find "recreation" in the tale. Assuming the semblance of an old diary, discovered by accident in this latter day, it permits its "editor" to adopt the leisurely style of the elder authors, and discard the modern method of eliminating every character and event which does not actively forward the development of the plot.

Plot there is, to be sure, and plenty of it, including battle, murder and sudden death, but generously mingled with it are amusing character studies, professional dissertations, careful delineations of the life of the time, with especial reference to the state of medical science, hints at social and political problems, all bound together by the indispensable love story, culminating in the "lived happy ever after."

The villain of the piece has pathological reasons for his fiendish performances; the hero slays his would-be murderer with his lancet, and in short the whole story is strongly colored with the profession of its author, who has evidently enjoyed himself immensely in the construction of the work, and may safely expect that many of his brethren will find equal enjoyment in reading it, while their patients, even if inclined to skip the strictly medical portion, follow the exciting chain of incidents and laugh at quaint remarks of woodsman and innkeeper. Altogether the volume is a worthy successor of the two that have gone before.

Jelliffe.

A Compend of the Practice of Medicine. By Daniel E. Hughes, M.D., Late Chief Resident Physician, Philadelphia Hospital; Late Physician in Chief, Insane Department, Philadelphia Hospital; Formerly Demonstrator of Clinical Medicine in the Jefferson Medical College of Philadelphia, etc., etc. Seventh Revised Edition. Edited, Revised, and in Parts Rewritten by Samuel Horton Brown, M.D., Assistant Dermatologist, Philadelphia Hospital; Assistant Dermatologist, University Hospital Dispensary, etc. P. Blakiston's Son & Co., Philadelphia.

The reviewer needs add but little to the information conveyed by the title page of this volume. Hughes' Practice of Medicine is a very handy volume, and the name of Dr. Brown is sufficient guarantee for the additional features of the new edition, especially the remarkably complete section on Dermatology.

The modern divisions of pathology have dictated the rearrangement of the diseases, and the prescriptions and modes of therapy in general have been thoroughly modernized. In the section on Mental Diseases the rather colloquial style of the previous editions still appears to a considerable extent, but elsewhere up-to-date methods of expression have been adopted and the valuable matter of the book retained in this new dress.

Much new material has been added, as the articles on the classification and general characteristics of fevers, examination of the blood, sputum, urine, and the like. The whole work is exhaustively and carefully indexed, and the book is gotten out in the characteristic style of this publishing house, which imparts to the most severe text-book something of the appearance of an edition de luxe.

Goodale.

Histologische und histopathologische Arbeiten über die Grosshirnrinde mit besonderer Berücksichtigung der pathologische Anatomie der Geisteskrankheiten. Herausgegeben von Frantz Nissl, Professor der Psychiatrie in Heidelburg. Vol. I. Gustave Fischer, Jena.

The contents of this volume bear excellent testimony to the intellectual activity that in the main characterizes the work of the medical officers in the German phychiatrical clinics and that leads to the printing of the results of important investigations, not only in the various journals or archives, but in more or less independent publications. The Preface, written approximately at the same time that Nissl assumed the direction of the Heidelburg Clinic, a position made vacant by Kraepelin's acceptance of the professorship in Munich, seems to indicate the lines along which the work of the investigation is to be prosecuted under this new régime, and the program may be safely adopted by all workers in this field. We are told that the knowledge required to-day of symptoms or structural changes will indicate the line of advance on the day following. There are to be no jumps in the dark, and every effort will be made in attempting to further special lines of investigation to do so in a manner that will render the present technique most effective. An effort will be made to study accurately and in detail all histopathological changes occurring in the cerebral cortex and to endeavor to interpret their significance. The fact that up to the present moment investigators have only succeeded in demonstrating the existence of lesions of unknown significance in the brain should be an incentive, not a detriment, to the carrying out of further studies. The investigator is reminded that in dealing with these and similar problems in the laboratory, questions of biological importance are to be solved; and enthusiastic but inexperienced and poorly-trained observers are cautioned against attempting to interpret the nature of diffuse lesions in the parenchyma; for example, those occurring in catatonia, until they become familiar with the alterations that exist in focal lesions, such as areas of softening cysts, etc.

The first one of the two contributions in this volume is by Alzheimer, and discusses the possibility of the differentiation of the histopathological changes in the cortex in dementia paralytica from those found in other conditions. In a few prefatory remarks the important service rendered to the clinician by the pathologist in the study of paresis is briefly recalled, and the writer proceeds to discuss the relative value of the macro- and microscopic lesions in the establishment of the anatomical diagnosis. Under the first head a number of interesting observations are recorded; as for example, the fact that in 13 cases out of 170 the bones of the cranial vault were normal, or the complete absence of hydrocephalus externus in 19 instances, and the non-existence of cortical atrophy in the brain of one patient who had suffered from marked symptoms of the disease for at least four and one-half years. Among the meningeal lesions those affecting the pia were constant and in a measure characteristic, consisting in an infiltration of plasma and "mast" cells, as well as lymphocytes, accompanied now by progressive and again by regressive tissue alterations in the vascular system of either a proliferative or degenerative nature, and involving the endothelial anl connective tissue elements of the pia. The vascular lesions in paresis may be clearly distinguished from those occurring in arterio-sclerotic or hyaline degenerations, and may be said to include the growth of endothelial elements, the tendency to the formation of new vessels by budding, the vascularization of the intima, increase of elastic tissue, dilatation of adventitial lymph spaces, and the occurence of plasma cells in the infiltrate in every case, even in the most acute forms of the disease. The regressive changes may lead to occlusion of vessels and hyaline degeneration. The so-called rod or sausage cells (Stäbchenzellen) are constantly present. Alzheimer's careful description of these elements in their most characteristic as well as in their transitional forms, and illustrated by a series of figures in the plates, forms by itself an important contribution to the pathology of the central nervous system.

There are no specific alterations in the nerve cells, although many evidences of the great severity of the process affecting them exist, and may be described as a form of necrobiotic degeneration. The early degeneration of the medulated fibres, as well as of the finer neural structures, which are interposed between the ends of the medullary sheaths and the ganglion cells is eminently characteristic. The increase of the glial elements and their structural anomalies are discussed in detail. Although the subject of localization of the paretic process receives careful consideration, the author makes no attempt to explain why certain parenchymal areas are more severely affected than are others, but the opinion is expressed that, after all the facts are carefully weighed, it is impossible to regard the changes as the result merely of a vascular lesion.

The more or less complete involvement of the parenchyma of the brain by an inflamation essentially different from other inflammatory conditions that attack this organ seems to be the cardinal point that distinguishes dementia paralytica from all other organic diseases of the central nervous system. The pages devoted to the description of the distinguishing characteristics of the paretic, alcoholic, syphilitic, and arterio-sclerotic processes contain information of equally great interest to the clinician and pathologist. The illustrations in the text, as well as the plates, add very materially to the value of the monograph, and deserve careful study by all those who are interested not only in the investigation of this particular disease, but equally by those who desire to gain an idea of the advances made within recent years in the study of the histology and pathology of the cerebral cortex.

We believe that there is a reason to justify the affirmation that this contribution of Alzheimer is the most important one made within the last decade to the study of dementia paralytica.

Under the head of the histopathology of the disease process affecting the cortex in paresis Nissl discusses the question of the possibility of establishing the anatomical diagnosis of dementia paralytica in a given case without reference to the facts contained in the clinical record. Klippel's affirmation that paresis is a term used to indicate a clinical syndrome developing as the result of a variety of essentially different lesions in the cerebral cortex is categorically denied.

In regard to the mooted question of the relation of the paretic, arterio-sclerotic and syphilitic process Nissl takes issue with Staub, who, as the result of his observations, affirmed that it was possible to demonstrate so-called destructive syphilitic changes, in the sense in which they were defined bv Heller and Dochler, in the aorta of individuals who had succumbed to paresis; and also that the aortitis syphilitica could be definitely distinguished from the sclerotic process. The important bearing upon this controversy that such experimental work as that of José and Walter Erb, who produced an atheroma of the aorta in rabbits by repeated injections of adrenalin, has, is referred to. A wider experience has caused the writer to revise his previously expressed views to the effect that cases of paresis occurred in which there were no arteriosclerotic changes in the intima of the aorta or cerebral vesicle. The presence or absence of "rod cells" is a fact of great diagnostic importance, and the probable glial origin of these elements receives further confirmation. In the study of both progressive and regressive changes the distinctive morphological features that serve to distinguish the so-called typical "rod cells" from other cells which are not nerve cells, but are of ectodermal origin, becomes more and more apparent. The diagnostic importance of the presence of lymphocytes and plasma cells in the advential coats of the vessels is reaffirmed. The recognition of the fact that the so-called perivascular lymph space of His and Obersteiner's pericellular canal were not true lymph channels marked a decided advance in the knowledge of this disease and made it possible to differentiate more accurately between the various nuclei in the adventitial and extravascular spaces. The origin and pathognomonic significance of the plasma cells are subjects that are discussed at length. At present Nissl affirms that these elements are hematogenous in origin, and represent transformed leucocytes, this change taking place in the vessel walls. These cells exhibit a marked tendency toward regressive changes. If, after careful search, they are not found in the central nervous system, it is safe to infer that the paretic process does not exist; but on the other hand, their presence alone is not sufficient to justify the establishment of the anatomical diagnosis of dementia paralytica.

Havet's attempt to prove the relative unimportance of the plasma cell as a pathognomonic sign is said to have failed; while Mahaion's affirmation to the effect that it is impossible to distinguish clearly between diffuse cerebral syphilis and this disease is also discredited. The fact that the progressive and regressive parenchymal changes are attended by an involvement of the blood-vessels of an exudative nature, including plasma cells as well as those resembling lymphocytes, definitely establishes the view which regards the disease as of an inflammatory character.

Stewart Paton.

Studien uber motorische Apraxie und ihr nahestehende Erscheinungen; ihre Bedeutung in der Symptomatologie psychopathischer Symptomen komplexe. Von Dr. Arnold Pick. Verlagsbuchhandlung von Franz Deuticke. Leipzig und Wien.

This monograph is divided into four sections, which deal respectively with (1) motor apraxia as a symptom in post-epileptic disturbances of consciousness; (2) motor apraxia in the course of progressive multiple lesions; (3) motor apraxia as an accompanying symptom of localized central disease; (4) the significance of the attention in the etiologie of motor apraxia and the relation of instrumental amnesia to it.

As the above outline of contents indicates, this monograph is an elaborate discussion of those forms of apraxia with which symptoms traceable to motor derangement are associated. The general scheme of Wernicke for aphasia is followed and a plan made for subcortical localization. A large number of cases are cited and their symptoms discussed at considerable length. The controversial and theoretical character of the monograph is such that its abstraction is quite impossible in a short space.

White.

Zur Physiologie der Spinalganglien und der trophischen Nerven sowie zur Pathogenese der Tabes Dorsalis. Von Dr. Georg Köster, A.O., Professor an der Universitat Leipzig. Wilhelm Engelmann, Leipzig.

Professor Köster presents a short monograph embodying the results of experimental studies on the cells of the spinal ganglia, the connections with the spinal cord, the results of cutting of the peripheral nerves, of the posterior roots, etc. He then takes up the trophic disturbances that result from these various experimental lesions, studies in great detail the pathological changes produced in the spinal ganglion cells as a result of the cutting of the peripheral nerves and of the posterior roots, and then shows the similarities that exist between the lesions thus experimentally induced and the pathological findings of locomotor ataxia.

As is well known this hypothetical standpoint relative to the underlying pathology of tabes is not new, but it is worked out in this paper with much zeal and patient effort, for twenty-six sections were practised, on cats, dogs and guinea pigs.

As a result of his section studies he finds that after section of the peripheral nerves there results a degeneration in the posterior roots in from sixty to seventy days. This shows itself in a partial breaking down of the medullary sheaths and an atrophy of all the posterior root fibers. The central stump of the peripheral nerve degenerates with atrophy of its fibers, and on the average a distinct breaking down of the medullary sheaths, which latter degeneration is apparent after several months. The distal end of the cut peripheral nerve degenerates completely in two weeks, but complete regeneration of the peripheral nerve is possible. After separation of the posterior roots there takes place—beginning about the third month, a distinct breaking down of the medullary sheath in the peripheral nerves, begining at the finer end branches. The sensory fibers of the peripheral nerve stem, excepting here and there distinct swellings, were simply atrophied, and near the ganglia few fibers with degenerating sheaths were to be found. It should be interpolated that the breaking down of the medullary sheaths in the end branches of the sensory nerve following section of the posterior roots is considerable, and distinctly comparable to the degeneration in the posterior roots after section of the peripheral nerves. The central portion of the posterior roots degenerates in all its parts after a few weeks. the ganglionic portion degenerates after a few months deep into the ganglion itself, and there results a complete destruction of all the fibers in contrast to the results obtained in the degeneration of the central stump. A functional renewal of the cut posterior roots does not take place. As to tabes the author argues exactly analogous findings. Jelliffe.

News and Notes

New York Neurological Society.—The following officers were elected for 1905: President, Dr. Joseph Fraenkel; First Vice-President, Dr. J. Arthur Booth; Second Vice-President, Dr. Smith Ely Jelliffe; Recording Secretary, Dr. J. Ramsay Hunt; Treasurer, Dr. G. M. Hammond; Corresponding Secretary, Dr. F. K. Hallock; Councillors, Dr. B. Sachs, Dr. Adolf Meyer, Dr. Joseph Collins, Dr. Pearce Bailey, Dr. E. D. Fisher.

Article V. of the By-Laws, governing the nomination and election of officers:—

1. Nominations of officers shall be made at the meeting next preceding the annual meeting, and their election shall be by ballot.
2. The Recording Secretary shall cause the names of all the nominees for each office to be printed on slips, two copies of which, together with a copy of this By-Law, shall be sent to each member with the notice of the election.
3. Vacancies may be filled at any time by nomination at one meeting and election as in Section 1 of this article.

Dr. Pasquale Penta, Professor of Criminal Anthropology in the University of Naples, died in that city November 29, 1904, being still a young man. Professor Penta had identified himself with this branch of anthropology for many years. He edited a review, *The Revista di Psicologia forense e Anthropoligia*, now in its seventh year, which contains many of his contributions to anthropological science.

Private Institutions for Epileptics.—It seems not a little strange that there should be so many private hospitals for the care and treatment of the insane in this country, and practically none for epileptics. The reason for this is undoubtedly to be sought in the fact that the hospital care and treatment of the epileptic was almost totally neglected up to a few years ago, when Ohio, Massachusetts, New York and one or two other States began to make provision for them, but for the dependent class only.

We still greatly lack private hospital facilities for the better classes of epileptics, whose treatment away from their homes is desirable. It is now very generally conceded that before we can get the best results in the treatment of this obstinate disease, the individual who has it must submit to the closest medical care and attention for two or three years. It is exceedingly difficult to treat the epileptic satisfactorily in his own home. It is impossible to control him at home the way in which he must be controlled to make possible the best results. This fact is apparent not only to the general practitioner but to the neurologist, and particularly to physicians connected with special institutions in which epileptics alone are cared for.

We note that the Health Resort Company, of Rochester, N. Y., as indicated by an announcement in the present issue of this journal, is about to establish in "Glenwood," at Dansville, N. Y., a place for the care and treatment of epileptics. It is gratifying to learn that the managers of "Glenwood" have secured the services of so able a man in the treatment of epilepsy as Dr. James William Wherry, formerly connected with the Clarinda State Hospital, at Clarinda, Iowa. Dr. Wherry's writings on epilepsy give him a prominent position among the epileptologists of the country, and under his management "Glenwood" should take a place of value as the first private institution in this country solely for this greatly afflicted class.

Among the consultants we note the names of Drs. James W. Putnam, Edward B. Angell, Roswell Park, John W. Whitbeck, Charles Cary, George M. Gould, Matthew D. Mann, Adolph Meyer and William P. Spratling.

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Vol. 32 April, 1905 No. 4

THE

Journal

OF

Nervous and Mental Disease

Original Articles

BILATERAL CERVICAL SYMPATHECTOMY FOR THE RELIEF OF EPILEPSY, WITH REPORT OF THREE CASES; NOTES ON THE PHYSIOLOGIC EFFECTS OF CUTTING THE SYMPATHETIC, AND ON THE HISTOLOGIC CHANGES FOUND IN THE CASES IN QUESTION.*

By William P. Spratling, M.D.,
MEDICAL SUPERINTENDENT OF THE CRAIG COLONY FOR EPILEPTICS OF SONYEA, N. Y.
AND
Roswell Park, M.D.,
OF BUFFALO, N. Y.
PROFESSOR OF SURGERY, UNIVERSITY OF BUFFALO, ETC.

Cervical sympathectomy as a therapeutic measure for the possible relief of epilepsy has been tried in enough cases to create a fairly extensive literature on the subject. In 1902, Winter collected all cases operated upon to that date, including nine of his own, and his extensive consideration of the subject comprising 203 cases, in which he gives the results in the form of a table, together with 5 cases reported by Hopkins[1] in March of the present year, makes it unnecessary to review all the literature up that time.

According to Winter, 6.6% of the 122 cases that were well observed were cured; 13.9% of them were "preliminarily"

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* Read before the New York Neurological Society, Dec. 6, 1904.

[1] S. D. Hopkins, "A Preliminary Report of Bilateral Excision of the Superior and Middle Cervical Sympathetic Ganglia in Five Cases of Epilepsy." New York Medical Journal and Philadelphia Medical Journal, March, 1904, p. 448.

WINTER'S TABLE.

		Number of Operations.	Cured.	Preliminarily Cured.	Improved.	Without Success i.e., No Result.	Dead.	Total Well Observed Cases.	Without Further News.
1.	Baracz	1	..	..	..	1	..	1	
2.	Alexander	24	4	2	3	13	1	23	1
3.	Jaksch	2	..	2	..	..	..	2	
4.	Kuemmel	1	..	..	1	..	..	1	
5.	Bogdanik	1	..	..	1	..	..	1	
6.	Pean	1	..	..	..	1	..	1	
7.	Jaboulay	16	..	1	4	10	1	16	
8.	Jonnesco	96	4	8	7	11	3	33	63
9.	Minin	1	..	..	..	1	..	1	
10.	Bayer	1	..	..	..	1	..	1	
11.	Chipault	26	..	3	..	..	..	3	23
12.	Donath	3	..	..	..	3	..	3	
13.	Schapiro	1	..	..	..	1	..	1	
14.	Richard	1	..	..	..	1	..	1	
15.	Potemsky-Sciamanna	7	..	..	1	6	..	7	
16.	Bourneville	1	..	..	..	1	..	1	
17.	Deschamps	2	..	..	..	..	..	..	2
18.	Souques	1	..	..	..	1	..	1	
19.	Dejerine	1	..	..	..	1	..	1	
20.	Braun	9	..	..	3	4	2	9	
21.	Mariani	8	..	..	2	6	..	8	
22.	Winter	9	..	1	1	5	..	7	2
		213	8	17	23	67	7	122	91

cured, 18.9% improved, 54.9% not improved, while 5.7% died.

We interpret the term "Preliminarily" cured as used by Winter to include cases in which the earlier effects of the operation were favorable, but in which the cases were not kept under observation long enough to permit a positive statement of a permanent cure.

Something might first be said on the rationale of this therapeutic procedure as based on anatomical and physiological facts:

The effect of the operation may be twofold: 1st, by cutting off a certain amount of sensory stimulation from the viscera, i.e., preventing these stimuli from reaching the brain; 2nd, by influencing directly the circulation of the brain by changes in the caliber of the blood vessels through the action of the vascular nerves.

The sensory functions of the sympathetic are much less known than are its motor functions, and for that reason it would be difficult to say in what manner the resection of the cervical sympathetic would affect the influx of visceral stimulation to the brain.

On the second point more definite data can be given. Huber[2] found two kinds of nerve fibers in the pia mater of the brain, medullated and non-medullated. He considers the former to be sensory. The non-medullated ones he considers to be vaso-motor nerves forming primary plexuses in the adventitia. Through frequent branches of the fibers of the latter, an interlacing network is formed. Within this plexus is formed a second plexus not so well defined, lying evidently internal to the muscular coat and giving off terminal fibrils in the muscular tissue of the vessels.

While there is thus established a histological basis to the theory of the presence of vascular nerves for the blood vessels supplying the brain, this theory is also apparently confirmed by physiological experiment.

Lafforgue[3] observed in rabbits as a direct result of stimulation of the cervical sympathetic nerve (peripheral stump) spasmodic ischemia of the whole cerebrum, cerebellum and oblongata.

Jonnesco,[4] making his studies on 15 epileptics on whom section or resection of the cervical sympathetic, unilaterally or bilaterally, was performed, concludes that the cervical sympathetic contains *vaso-constrictor* fibers for the *head,* face, and heart, made evident by excitation with strong electric currents; that it contains, furthermore, vaso-dilator fibers for the inner part of the cheeks and lips, for the gums, inner and lateral part of tongue, *and for the brain,* brought out by the application of feeble currents. Empirical facts have, of course, precedence over theoretical considerations, and if cervical sympathectomy would lead to a prepondering percentage of cures in epilepsy, or even improvements in epilepsy, we would have to accept the fact, no matter what its explanation might be. Nevertheless, we should always endeavor to explain facts, and this is particularly desirable in the case in view because of the circumstance that the percentage of cases benefited by the operation is by no means such as to warrant an unqualified justification of the operation in epilepsy.

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[2] "Observations on the Innervation of Intracranial Vessels." Jour. of Comp. Neurol., 1899, Vol. 9, No. 1.

[3] Lafforgue, E., "Rech.. exper. sur l'action du grand symp. cerv. dans la production de certains accidents syncop. (syncopes reflexes)." Indep. médicale, 1897, p. 409.

[4] Jonnesco, "Physiologie du sympathique cervical," XIII. Congr. Internat. de Med. Section d'Histologie et d'Embryologie, 1900. Paris, 1901, comptes rendus, pp. 26-38.

Jonnesco and Floresco, "Physiologie du nerf sympathique cervical chez l'homme," Rapport par M .Franois Franck. Bull. de l'Acad. Medicine, 1900, 3eme s., Vol. XLIV., pp. 213, 214.

While the three cases we have to report will not add much to the casuistry of the operation in the way of percentage, the report may be of value from the fact that one of the patients presented an additional condition, a pronounced "tic" of the head and right arm, which in some manner we are unable to explain at this time, was radically improved by the operation in a way that seemed a year later would be lasting. A second factor in our cases was a thorough histological investigation of the parts of the sympathetic nerves that were extirpated. So far as we have been able to ascertain this has never before been done in cases of epilepsy.

POINTS IN EXCISION OF THE CERVICAL SYMPATHETIC BY DR. PARK.

In attacking the cervical sympathetic, it is worth while to remember that it contains dilator fibers for the pupil, motor fibers for the involuntary muscles of the orbit, vaso-constrictor fibers for the vessels of the head, secretory fibers for the salivary glands, and the accelatory fibers for the heart. There is perhaps no more mixed nerve in the body than this.

Its upper end may be recognized by its gangliform enlargement. The middle ganglion lies upon the inferior thyroid artery which is just below the carotid tubercle, i.e., the anterior tubercle of the 6th cervical vertebra.

The nerve may be best exposed by incision along the posterior border of the sterno-mastoid. It should be divided between its fibers, close to its inner side, in order to reach the inner fascia and to separate it from the deeper muscles after this fascia is divided. It must be separated close to the base of the skull where the upper ganglion will be found lying on the inner side of the anterior tubercle of the transverse processes of the second and third vertebræ and upon the muscles which are covered by the above fascia. Here, the lower end of the nerve having been identified, it should be caught with forceps and freed up to the base of the skull. Then the nerve trunk may be separated downward. The balance of the operation may be completed by continuing the above incision or by making another, beginning 1 cm. above the clavicle, along the posterior border of the sterno-mastoid and extending it upward 5 or 6 cm. The posterior border of this muscle must be exposed and separated upwards till the fingers meet in the tunnel thus made beneath it. The wound must be widely separated, the muscle drawn upward and forward, the inferior thyroid artery identified, and the middle ganglion found wherever it rests, which is usually inside the curve of the vessel. Sometimes this ganglion is replaced by a plexus, or the main branch of the nerve may pass behind the vessel. The nerve trunk should be caught at this point, and pulled upon so as to surely identify it with the cord still remaining in the upper wound. The identity having been made clear, the nerve trunk is loosened and drawn forward. The artery is freed from any plexus round it. Should one be operating upon a case of exophthalmic goiter it would be particularly desirable to separate and destroy the fibers of the middle cardiac nerve, which extends round and passes to the inner side of this thyroid artery. The nerve trunk, being drawn down beneath the artery, is followed outward and downwards to its lower ganglion where it lies behind the clavicle on the neck of the first rib, between the scalenus anticus and the longus colli. Care must be taken to avoid the vertebral vein which covers it.

The nerve trunks should be separated, if possible, from the efferent and afferent branches, and the vertebral artery should then be exposed, freed and drawn outward.

In goiter cases it is especially necessary to divide the cardiac branches, which form the lower cardiac nerve, as well as all the vertebral branches. After effecting all this separation the nerve trunk is drawn upward, loosened by gentle traction, and finally removed with the lower ganglion. This ganglion is exceedingly hard to expose in some necks. In cases of epilepsy it is perhaps not so necessary that this should be removed as in cases of goiter where it is the cardiac sympathetic which one is most anxious to influence.

LIFE HISTORIES OF CASES OPERATED UPON.

CASE I. A. S., male, aged 24 years; native of New York, single, and a clerk by occupation.

Family History.—Father and mother living, aged 46 and 41 respectively. Father had convulsions when younger, which were claimed to be "uremic." Mother has frequent headaches with "a feeling of weight in the head." A brother of 8 years has chorea. Maternal grandparents died at ages of 48 and 46 respectively, the cause of death in both being apoplexy.

Personal History.—Patient is first in line of birth of six children. He was born at full term after instrumental delivery. Mother reports that he received "slight cuts over cheek bones" at this time. He was breast-fed and began teething at the ninth month. Had "night terrors" during childhood and began to walk at the nineteenth month. He was subject to frequent attacks of epistaxia without apparent cause. When 8 months old he fell from a chair, striking on his face. The first epileptic convulsion appeared when he was 15 years old, the supposed cause being fright. A second attack occurred a week later. His aura consists of severe headaches and "spots before the eyes." A ball of fire, he states, appears "between his eyes," then "long lines of color" are seen and everything "appears to pass from left to right." Diplopia occurs and the seizure begins. He has marked convulsive movements of the face and right arm, which have been present since the age of 8 or 9 years. They are choreic in form and become more pronounced previous to a seizure, and they are especially marked when he becomes excited. He was admitted to the Colony in June, 1903. He had one seizure in that month, one in August, two in September and two in November.

He was operated on by Dr. Park on November 16, 1903, and had a seizure the day after the operation. After that they ceased entirely until the following April—a period of four months—when he had two, after which they again ceased and have not recurred to this time, a period of seven and one-half months.

Operation and Clinical Notes.—Examination made just before the operation on November 16, showed the following: Tall, well nourished man of 24; ruddy face, red ears.

Pupils equal, slightly wider than normal. Reaction to light sluggish, response to accommodation paradoxical, i.e., dilation instead of contraction.

Choreic Movements.—Sometimes under excitement, and again without apparent cause, patient exhibits twitching movements of the face and head. The twitchings are usually not symmetrical, usually more marked on right side. Sometimes there are only one or two, again a longer series in quick succession, clonic like, at other times they are chiefly tonic. In the face the twitchings often appear as grimaces, distortions of different kinds. They are at times accompanied by a coughing sound.

Arms also affected to some extent. Here the movements have a more choreic character (as they have to less extent also in the other parts affected.) *Deep reflexes of upper extremities, elicitable,* but weak, equal on both sides, *knee jerks* equal, of good strength, but not lively; *ankle jerks* both present, equal, no ankle clonus; *plantar reflex* very weak on both sides, of Babinski type on left; normal type on right. *Heart action* not increased in strength. Heart sounds all clear. No exaggeration of second aortic sound. Pulse slow, average beat, 53, but decidedly arythmic. From five to ten slow beats are followed by two or three rapid ones, so that if the slow ones alone were counted, the slowness of pulse would be still more apparent. The accelerations of the pulse are frequently concomitant with the twitching, although not always so. Pulse is rather full, though changing in tension. *Respiration* on the whole, slow, about 13, but it is influenced by the choreic twitchings, which affect also the respiratory muscles, making respiration at times faster and deeper and superseding sometimes the normal respiratory movements so as to cause two instead of one distinct muscular contraction in one phase. *Lungs*—Some wheezing, particularly on the right side, lower part; otherwise normal. *Legs* show motor restlessness, reminding of chorea, but the movements in them have less of a spasmodic character. *Hands* show no tremor. *Thyroid* not enlarged. *Liver* and *Spleen* not enlarged. *Abdomen* rather retracted and dense.

Both cervical sympathetic nerves were removed by Dr. Roswell Park, November 16, 1903. Both nerves were found accompanied by a chain of lymphatic glands, the surroundings of which show increase of connective tissue, causing numerous adhesions, so that it is difficult to reach the sympathetic nerves. The latter, themselves, are strongly involved in the adhesions.

After section of the first cervical sympathetic (left), the pulse became very bounding; after the removal of the second it became still more so, but the difference was not so marked as after the removal of the left alone.

Both sympathetics preserved in 6 per cent. formaline immediately after removal. Apparently most of both upper cervical sympathetic ganglia and both middle cervical ganglia were removed and parts of the nerve below the latter.

Pupils became contracted while patient was still deeply anesthetic under ether.

The pulse became much less full by the time the patient was removed from operating table.

November 17.—Patient vomited once after operation. Did not sleep during night. *Face* of same color as before operation, perhaps slightly more congested. Conjuctiva slightly injected. *Pupils* both markedly contracted, but equal. Well marked ptosis on both sides. Pulse 68, rather bounding, but considerably less than directly after removal of sympathetic. Apparently less choreic twitchings before operation.

November 18.—Pulse about 120, but so erratic that it is hard to count it, and especially since every movement of patient gives a marked acceleration. Had an epileptic attack this morning, 9 :40 Half hour later, restless and confused. Speech thick and blurred; one can hardly understand what he says.

November 20.—Complains of numbness of left ear and region behind ear, also of numbness under the chin. Same symptoms existed to less extent on right side, but have now disappeared. The numbness appeared after the operation.

The posterior surface of left ear and an area behind the latter, about one inch wide, show impairment of tactile sense.

Knee-jerks with Jendrassik rather subnormal, sluggish.

The patient made a rapid and satisfactory recovery from the operation.

Histological Notes (Case A.S.)—The two cervical sympathetic nerves in this case were put in 6 per cent. formaline immediately after removal, and their histological examination gave the following results:

Left Nerve.—Transverse sections stained after Busch showed no change. No inflammatory or vascular changes.

Right Nerve (below superior cervical ganglion).—Weigert and Pal sections, some cut transversely, some obliquely, show the medullated fibers normal for the most part.

Some small areas are unstained, but as these differ in different sections they are probably artefacts. Only few fibers show irregularities of caliber. Busch sections (longitudinal) are negative.

Below the upper cervical ganglion the nerve seems to consist almost entirely of medullated fibers.

Right Superior Cervical Ganglion.—Busch sepecimens (unstained or counterstained) show pigmentation of nerve cells chiefly in the peripheral zones of the cells otherwise negative. No inflammatory or vascular changes. Cells show no atrophic changes whatever.

Right middle cervical ganglion shows an inflammatory focus with tortuosity and dilatation of vessels.

Nissl specimens of this ganglion show too much artificial shrinkage (due probably to over-heating during the process of paraffine embedding) to allow very definite conclusions. The cells, however, seem normal so far as structure of the stained substance (Nissl bodies) is concerned.

The medullated nerve fibers of this ganglion show rather even caliber and are apparently normal.

Plate I

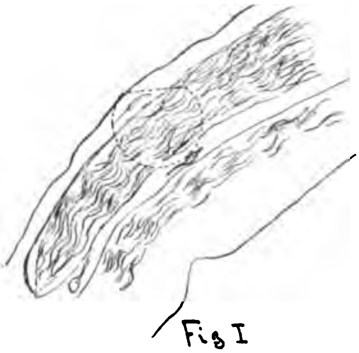

Fig I

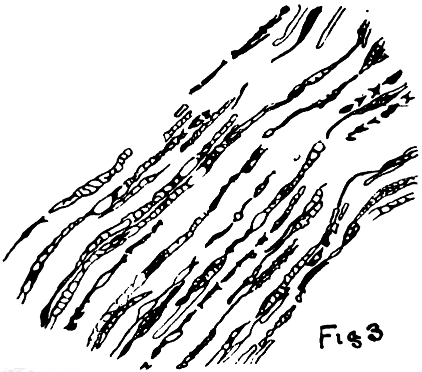

Fig 3

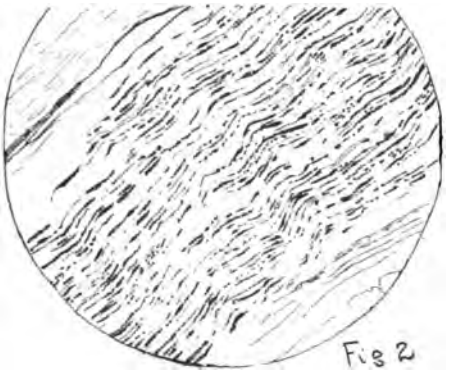

Fig 2

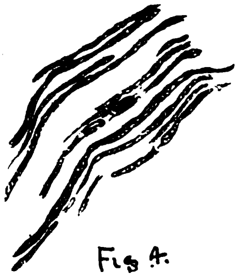

Fig 4.

Plate 2.

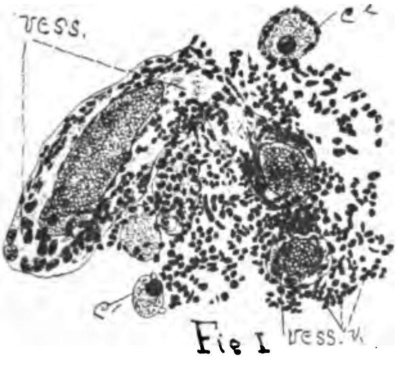

Fig I

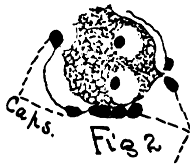

Fig 2

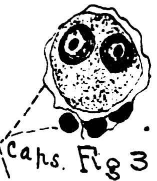

Fig 3

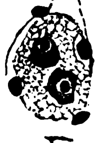

Fig 4

Left upper cervical ganglion: Nissl specimens show only a few cells pigmented. Relatively slight shrinkage, evidently artificial, since celloidine sections show no such shrinkage.

Chromophile structure apparently normal. In the specimens from which the sections stained with Bohmer hematoxylin and eosine and the sections stained after Van Gieson were taken, pigmented cells are more numerous, but also not a great number.

Nerve fibers in Wolters-Weigert specimens show marked irregularities in caliber, and seem rather scarce (?).

Left middle cervical ganglion: Nissl specimens present nothing abnormal except on one section a cell with a double nucleus and the presence of numerous pigmented cells. The cell with the double nucleus is illustrated in Plate II., Figure 2;—caps. is the surrounding capsule of the cell with nuclei embedded in it.

Busch specimens also show pigmentation of a number of cells.

Case II. (H. B.)—Age twenty-two years; no occupation, single.

Family History.—Both parents living. Mother subject to headaches; father a moderate drinker; a maternal aunt is insane; maternal grandfather, rheumatic. Maternal grandmother died of "tumor," paternal grandfather of cancer, and paternal grandmother of heart disease.

Personal History.—Patient is fourth in line of birth of five children. Normal birth at full term; said to have been a strong child; teething commenced at the 8th month. No history of spasms or convulsions at the time; commenced to walk about the 15th month.

First epileptic seizure occurred at the 18th month when she had a series of them; the cause being unknown. Aura consists of vertigo with marked flushing of face. Has a left hemiplegia, indistinct in character, and probably due to a cerebral hemorrhage that was caused by the first series of convulsions.

She was admitted to the Craig Colony in May, 1902. She had an attack that month; one in July and five in October following. During the next year she had four attacks only. She was operated on by Dr. Park on November 16, 1903.

The left sympathetic nerve was removed comparatively easily. No such chain of lymphatic glands and adhesions being present here as in the cases of A. S. and G. S. operated upon to-day.

After removal of the nerve, pulse became fuller and went down from 108 to 84.

On the right side a piece of nerve was removed, but the fact that it has no distinct ganglioniform enlargement makes it very doubtful whether it is the sympathetic. This doubt becomes confirmed by the fact that histologically no nerve cells could be made out in the resected piece, and is also clinically confirmed by the absence of contraction of the pupil of that side after the operation, furthermore, after the removal of this second nerve the pulse did not become fuller than it was after removal of the left sympathetic.

November 17.—Patient shows ptosis of the left upper lid, contraction of left pupil and some injection of vessels of lower half of left conjunctiva.

Right pupil not contracted. No ptosis on right side. Face is not more congested than before operation. It is evenly and strongly reddened on both sides.

Histological Notes (Case H. B.)—The removed left sympathetic was put in 10 per cent. formalin immediately after removal.

It proved to consist only of the left superior cervical ganglion plus the cord below, without the middle cervical ganglion.

The piece resected on the right side was not the sympathetic, but some other nerve.

Left superior cervical ganglion: The Nissl specimens are considerably shrunken owing to over-heating during paraffine embedding. While this makes interpretation difficult, the chromophile structure of the cells was apparently normal.

A fair number of cells show pigmentation. Three cells show a double nucleus, one apparently a triple nucleus, although the latter structure is somewhat doubtful of interpretation. No inflammatory changes found, no vascular ectasies.

Left nerve below superior cervical ganglion: Weigert specimens were lost.

Busch specimens showed no changes.

Case III. (G. S.)—Female, age twenty-six years, native of New York, housewife.

Family History.—Father living, aged seventy years. Mother died at age of forty-four years of typhoid fever. Paternal grandparents died at ages of seventy-three and eighty years respectively. Maternal grandfather died of tuberculosis. Maternal grandmother at age of ninety years. A paternal aunt had epilepsy. Mother had "fainting spells" and rheumatism. Father, rheumatic.

Personal History.—Patient was fourth in line of birth of five children. Born at full term after a normal labor. Delivery normal. She was a strong child, and began teething about the 8th month. No spasms or convulsions during first dentition. She had measles at the age of five, and diphtheria at six years. At age of seven years she fell and "bumped" her head on a table. When four years old, she had an attack of cholera infantum attended by convulsions, which disappeared on recovery from the cholera infantum. The first genuine epileptic attack is said to have occurred at the seventh year, during convalescence from typhoid fever. She was mentally disturbed during fever. Her attacks are preceded by vertigo and general numbness of body. Before admission to the Colony her attacks occurred from 3 to 10 days apart. During series of attacks her face becomes very red, she is feverish, and complains of prostration. She was married in her eighteenth year and has three children. No miscarriages. Lacerated cervix uteri. She has an aura about half an hour before her seizures in the nature of "gastric distress," and says this has been the case since the birth of her first child—ten years ago. Before that she experienced a "faint feeling" in the stomach ten minutes before the onset of the attack. She also complains that "her face burns like fire before attacks."

She was operated on by Dr. Park on November 16, 1903, and the day following the operation she had a double ptosis more marked on the left side. This condition completely disappeared in about two weeks.

Both nerves were found involved in numerous adhesions accompanied by a chain of lymphatic glands as in the case of A. S.

Left nerve was not preserved. Right one was found to be unusually thin. The resected piece contained the right upper cervical sympathetic ganglion and a piece of the nerve below.

November 17.—Patient has not vomited; slept about two hours and a half in the night.

Face is markedly congested, but patient says it was always red.

Ptosis of both upper lids, slightly more marked on right side. Pupils both equal and contracted, responding only slightly to light. Pulse 120, rather small; respiration 22. Pulse full and bounding.

Histological Notes (Case G. S.)—In this case only a piece of the right superior cervical ganglion and a piece of the nerve below were examined; the left cervical sympathetic was not preserved.

Right superior cervical ganglion: A considerable number of nerve cells of this ganglion show pigmentation in slight degree, and one nerve cell at least in the ganglion showed two nuclei very clearly, each with a nucleolus. The section showing it was stained after Van Gieson. (See Plate II., Figure 3, illustrating the cell with its two nuclei and with the surrounding capsule *(caps.)*.

Nerve.—The right cervical sympathetic nerve below the superior cervical ganglion showed some changes in its medullated fibers which at first glance seemed pathological in a marked degree. The medullary sheaths showed great irregularity of stain in sections stained after Wolters-Weigert or Wolters-Pal. An illustration of them is given in Plate I., Figures 2 and 3, both showing the same region of the nerve under different magnifying power. Plate I., Figure 1, shows diagrammatically the nerve in its entire thickness. The dotted line in this figure represents the entire area illustrated in Figure 2.

As it was suspected that the irregular staining might be the result of formalin hardening, the sympathetic nerve of an apparently normal sheep, also hardened in formalin, was used for comparison, and inspection of Plate II., Figure 4, will show that here too, the medullary sheaths show the same mottled appearance, which must, therefore, be attributed to the hardening. At the same time, it is noted that the nerve fibers of the normal sheep do not show the marked irregularities of caliber seen in the nerve of the patient and illustrated in Figures 2 and 3.

EPICRISIS OF THE HISTOLOGICAL CHANGES FOUND.

The changes found in the removed sympathetic nerves of the three operated cases are in essence as follows:

1. Pigmentation of a greater or less number of nerve cells of the cervical ganglia in all three cases.

2. Presence in every one of the three cases of at least one nerve cell with double nucleus in some one of the extirpated ganglia. In one of the cases about half a dozen such cells were found.

3. Degenerative changes in the medullated nerve fibers in the sympathetic cord and ganglia of the excised portion.

4. In one case (A. S.) a focus of inflammation, i.e., of perivascular round cell infiltration.

We will touch briefly upon the significance of the pigmented cells. It has been found in so many pathological studies that its true interpretation is difficult. Whether the constancy with which changes found in the nerve cells of senile people allows us to consider it as a sign of senility of the nerve cell, remains questionable in view of the fact that certain nerve cells (i.e., of the substantia nigra Soemmeringi and substantia ferrugine) are pigmented normally. Whether they also possess normal function, it is rather difficult to say before more is known on this point. The view that they might possibly represent a phylogenetic senility is purely speculative.

But a point of interest, warranting discussion, is the presence of nerve cells with two nuclei found in all three operated cases. It should be pointed out that in making this observation great care was taken not to mistake the nuclei of the capsule of the cells for intracellular nuclei or nucleoli, and, furthermore, not to mistake two closely adjoining cells for one cell with two nuclei. The "Nissl" stained cell with double nucleus gave a particularly clear, unmistakable picture.

But we must remember that in the sympathetic nervous system of certain mammalians, certain cells with two nuclei are not unusual. Schwalbe[5] has shown mononuclear as well as binuclear cells in the ganglia of the sympathetic chain of young rabbits. With advancing age the number of monuclear cells decreases in the same proportion to which the binuclear ones increase, so that the mononuclear cells are to be considered as a fore-stage of the binuclear. According to Apolant[6] and Schwalbe, the binuclear cells are found also in the large abdominal ganglia and in the smaller cardiac ganglia. Huber[7] found them in the solar ganglion.

Their frequent occurrence in the sympathetic nervous system even gave rise to an attempt to utilize this feature for assigning ganglia or sporadic cells to the sympathetic or cerebrospinal system respectively. Since, however, Key and Retzius[8] found occasional cells with a double nucleus also in spinal ganglia of rabbits, the binuclearity of the nerve cells cannot be considered as absolutely diagnostic of the sympathetic system.

In the sympathetic nervous system of man, cells with a double nucleus are evidently rare. This was pointed out by Brueckner[9] in a paper on "The Fine Structure of the Sympathetic Cells," published in 1898.

In the central nervous system of man, Brueckner found cells with two nuclei in healthy individuals as well as in a patient with typhoid fever, and in a piece of brain which Jonnesco extirpated in a case of Jacksonian epilepsy followed by recovery.

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[5] Schwalbe, "Uber den Bau der Spinalganglien," etc., Arch. f. mikr. Anat. Bd. IV., 1868.

[6] H. Apolant, "Ueber die sympathischen Ganglienzellen der Nager," Arch. f. mikr. Anat. Bd. 47, p. 461, 1896.

[7] G. Carl Huber, "A Contribution on the Minute Anatomy of the Sympathetic Ganglia of the Different Classes of Vertebrates," Journal of Morphology, Vol. XVI., No. 1, 1889.

[8] Key and Retzius, quoted by Apolant, l. c.

[9] Brueckner, Arch. des Sciences Médicales, Vol. III., 1898, p. 197.

In view of these facts, it is questionable whether the presence of binuclear cells in the removed cervical sympathetic of our three operated cases has any bearing, direct or indirect, on the epilepsy. For the present it is simply to be signalled as a fact that the binuclear cells were found in the cervical sympathetic in three successive cases in which these nerves were removed. Future observation will have to show whether such cells are not found with equal frequency in healthy individuals or in other pathological states.

The following observation still deserves notice in this connection: Obregia and Bresnia[10] after electrical excitation of leeches found that part of the nerve cells of this animal increased in size and presented all at once two nuclei which moved away from each other towards the two "extremities" (poles) of the cell. The latter begins to show a slight furrow, which becomes more and more accentuated until it finally leads to complete division of the cell. As they never observed a karyo-kinesis, they concluded that this multiplication occurs by simple division,* and explain in this manner also the regeneration of brain observed after experimental extirpation by Vitzu and Tedeschi.

These interesting facts certainly call for further experimentation and it would be premature now to offer any definite conclusions in regard to them.

As to the inflammatory focus found in the second case (Stern) operated on by Dr. Park, it is doubtful how much importance should be attributed to it. It was undeniably the most marked of all the changes found, and it corresponds to a degree, with the gross changes observed by Dr. Park in connection with the sympathetic, i.e., the adhesions of the nerve and swelling of the lymphatic glands accompanying it. But the question occurs as to whether these inflammatory changes were primary and whether the irritation of the sympathetic produced by them had any causal relation to the epileptic seizures, or whether on the contrary, the circulatory disturbances and changes of metabolism produced by the seizures themselves led secondarily to the inflammatory changes in and around the cervical sympathetic nerves.

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* It is appropriate to mention here that in the cervical sympathetic of the cases operated on by Dr. Park, no cells presenting signs of karyokinesis were found.

[10] Obregia and Bresnia: From Brueckner, l. c.

To decide this question the material was not sufficient. It is important that the sympathetic nerve be examined in cases in which it is removed for therapeutic purposes in epilepsy, and which, peculiarly enough, seems never to have been done to this time: at least so extensive a contribution to cervical sympathectomy in epilepsy as the paper of Winter,[11] that containing a discussion of 203 cases is entirely silent on this point; and Jonnesco,[12] who has done so much work along the same line, simply points out the "frequency with which lesions of the cervical sympathetic were found in association with ophthalmic goiter and epilepsy," without offering any substantiation to his statement. Indeed, our search of the literature for anatomical reports of lesions of this nerve in epilepsy has so far been entirely negative. Yet if cervical sympathectomy is so frequently resorted to as a therapeutic procedure in epilepsy, with a view of correcting circulatory conditions in the brain and the influx of visceral stimuli to the latter, it is but natural to eventually look for the cause of these anomalous conditions in the sympathetic nerve itself. In the light of the foregoing it would seem appropriate to histologically investigate the cervical sympathetic in such cases.

A few words in conclusion about the degenerative changes found in the medullated nerve fibers of the removed cervical sympathetics of the cases of G. S. and A. S. They were not very well marked and left some doubt as to their true interpretation. One conclusion to be reached is that preliminary formaline hardening is not favorable to the preservation of the medullated nerve fibers in the sympathetic, and should be supplemented by osmic acid teased specimens. The Busch modifications of the Marchi method was unsatisfactory. New methods, bringing out the fibrillary structure of the axis cylinder will eventually have to be resorted to, for investigating the condition of the non-medullated fibers of the sympathetic. They seem the only ones promising to throw light on the conditions of these fibers in pathological conditions, although the lack of reliability of most of them makes it doubtful whether they can be successfully applied for the purpose.

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[11] Winter, Archiv. fuer Klinische Chirurgie, 1902, p. 816.

[12] Jonnesco, Centralbl. fuer Chirurgie, Jan. 16, 1897, p. 33.

We express our appreciation of the very valuable services rendered us in the preparation of this paper by Dr. B. Onuf, the resident pathologist at the Craig Colony for Epileptics.

EXPLANATION OF PLATE I.

Figures 1, 2 and 3 represent a piece of the right cervical sympathetic nerve of case G. S.

Figure 1 illustrates the nerve diagrammatically in its entire thickness (magnified sixty-five times in diameter).

Figue 2 (magnified 300 times in diameter) represents the area enclosed in the dotted line of Figure 1.

Figure 3 shows a portion of the sympathetic nerve of an apparently normal sheep (under oil immersion, magnified 750 times in diameter).

EXPLANATION OF PLATE II.

Figure 1. Inflammatory focus in the right middle cervical sympathetic ganglion of case "Stern." vess.. bloodvessel; r., round cells; c', nerve cell; c', nerve cell with capsule.

Figure 2. Nerve cell with two nuclei, each nucleus with a nucleolus; taken from the left middle cervical sympathetic ganglion of case "Stern." Nissle stain (oil immersion, magnified 750 times in diameter); caps., capsule surrounding the cell.

Figure 3. Binuclear nerve cell from the right superior cervical ganglion of case "Grace Sheffield." Van Gieson stain (oil immersion, magnified 750 times in diameter); caps., capsule.

Figure 4. Binuclear cell from left superior cervical ganglion of case "Harriet Baker." Van Gieson stain (oil immersion, magnified 750 times in diameter); caps., capsule.

MYELOMALACIA, WITH ESPECIAL REFERENCE TO DIAGNOSIS AND TREATMENT.*

By F. W. Langdon, M.D.,
of Cincinnati.

Softening of the spinal cord, not consecutive to a myelitis, is practically always a sequel to arterial thrombosis.

It is an event of much more frequent occurrence than primary myelitis.

Early recognition of the nature of the process is of great practical importance, since upon such recognition depends the good effects of a rational therapy and consequently the best interests of the patient.

The foregoing propositions have been familiar to neurologists the world over for almost a decade, and have gradually gained in favor, until at the present day it is conceded that myelomalacia is a very frequent condition as compared with primary myelitis. In other words, a large majority of the cases on record as acute myelitis have been really cases of acute softening due to thrombosis and followed by inflammatory exudate around the softened area.

Williamson, in a remarkably comprehensive series of articles, (*Manchester Medical Chronicle*, 1894 and 1895) is usually credited with being the first to draw attention to the occurrence of softening in the cord of thrombotic origin, and to present actual demonstration of the thrombi in a case of myelitis as then recognized.

Bastian however (Quain's "Dictionary of Medicine," 1882 and 1885) had previously on purely rational grounds advocated the view that thrombotic spinal softening was a frequent event.

Singer (*Brain*, 1902. p. 332) has presented a very concise and satisfactory statement of the subject, and advocates the view that the "majority" of recorded cases of so-called "acute myelitis" are "not inflammatory" but due to thrombosis of spinal vessels.

While this frequency of myelomalacia and the corresponding rarity of true primary myelitis are generally recognized by neurologists, it is still an obvious fact that most of our standard text-books on neurologic medicine, even those written by eminent masters of the art, have continued to group both conditions under the label "myelitis."

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* Read at the meeting of the American Neurological Association, Sept. 15, 16 and 17, 1904.

In fact the term myelitis has about as much pathologic significance at the present day as the old term "apoplexy" in respect to brain lesions. It is not a pathologic diagnosis but merely a statement of loss of function in a given region of the nervous system.

Dana, for instance ("Text-book of Nervous Diseases," 5th Ed., 1901, pp. 249-250), states "It (myelitis) may be initiated or accompanied by a hemorrhage or softening from thrombosis of arteries, and the latter is often the case."

"*Nor can we clinically distinguish* between the cases due to a primary infection and those due to a hemorrhage and softening. Hence acute transverse myelitis may mean an acute inflammatory process or an acute softening." (Italics by present writer.)

Church and Peterson ("Nervous and Mental Diseases," 4th Ed., 1903, p. 351) consider "Thrombotic Softening of the Cord" under a separate heading. Here we find the statement that "Thrombosis of the arteries of the spinal cord undoubtedly occurs."

"This result has ordinarily been confounded with myelitis *and clinically presents the same picture.* (Italics by present writer.)

"As the *symptoms, treatment and prognosis are those of myelitis* it is not necessary to repeat them here."

Oppenheim ("Diseases of the Nervous System," Trans. by Mayer, 1900) includes under "Myelitis," "inflammations and softening processes" and further remarks "still, our conception of the term is not a precise one."

Even so discriminating an author as Gowers does not recognize myelomalacia as a clinical entity but speaks of "softening" only as a sequel of myelitis ("Manual of Nervous Diseases," Gowers and Taylor, 3d Ed., 1899).

Starr ("Organic Nervous Diseases," Phila., 1903) considers myelomalacia and myelitis separately, emphasizes the comparative frequency of myelomalacia and the rarity of myelitis, but states (pp. 347-348). "Whether a clinical distinction can be reached between these two types on account of the development in the infectious type of a marked febrile invasion with symptoms of an acute infection and leucocytosis, which are wholly absent in the second (myelomalacia) type, is a matter for further investigation."

The present paper is an attempt to establish on a practical basis, such a clinical differentation between the two diseases and to point out the principles of treatment appropriate to each.

DIAGNOSIS BETWEEN MYELOMALACIA AND PRIMARY MYELITIS.

Omitting consideration of questions of heredity and predisposition, as too remote for our present purpose, *the immediate prodromata* as a rule differ decidedly in the two conditions.

In myelomalacia the patient has commonly been attending to his customary work or duties, often with *no sign of illness before the onset* of the disease. There is no "chill." Pain is absent. There may be however transient attacks of numbness, tingling or sense of weight in one or more extremities, which are not of sufficient severity to prohibit his ordinary activities. Evidence of syphilis or other cause of vascular disease is commonly present.

In myelitis, on the contrary, the patient has been ill with some acute infection due to staphylococci, gonococci, pneumococci, etc., or one of the exanthemata is present. Traumatism, tumor or vertebral disease must of course be considered. In other words *the patient has been otherwise ill* aside from the cord disturbance proper.

The onset of myelomalacia is sudden as it is always in vascular nervous lesions whether the cord or brain be involved. There is no "chill"; pain is absent. Notwithstanding this suddenness of onset, the immediate loss of function may be slight in extent, perhaps a mere numbness or sense of weight in foot or leg, but it *persists* and *increases* often by additional *sudden increments* within a few hours or days. If these increments be absent, the symptoms, whatever their nature, remain stationary for a few days, perhaps a week and then show a period of extension or increase due to *exudative inflammation* around the softened area.

It is this secondary inflammation which often causes the patient to cease work on account of the motor weakness at this period.

Should the focus of softening, as is often the case, be confined to the irrigation field of one artery, say in an anterior horn, the resulting motor paralysis at the end of a week is still unilateral, while *marked sensory defects,* especially in recognition of heat and cold are present in the *opposite limb.*

In myelitis on the contrary the *onset is gradual.* Malaise, backache and "chill" often precede the sensory and motor symptoms; a convulsion may replace chill in the young. The primary cord symptoms are more commonly bilateral as well as more diffuse longitudinally. They steadily progress, (not by sudden increments) so that in a few days or a week, complete paraplegia is the rule.

Fever is commonly absent early in myelomalacia, and when it does develop later is due to some complication on the part of the intestinal tract or the bladder or to bed-sores.

In myelitis fever of moderate degree is commonly present at the start and if the cervical cord be involved a sharp rise of temperature to 105 or more is the rule.

As regards the transverse extent of the involvement of the cord, in myelomalacia this may be slight. Only one anterior horn area, of one segment, may be involved, one artery occluded. Sensory losses may be absent on the side of motor loss but marked in the opposite limb or area. This applies especially to temperature sensations. Such a "pattern" of functional losses means commonly, obstruction of a single anterior horn artery. But for its sudden onset it would simulate the "dissociation symptom" of syringomyelia.

In primary myelitis, an entire transverse section of the cord is involved within a few hours or days, and commonly several segments, as indicated by the diffuse character of the symptoms.

It is obvious that a general infection in the blood stream is not likely to be confined in its distribution to one artery.

Looking at the individual symptoms, we may consider: (1) Sensory defects, (2) motor paralysis, (3) changes in reflexes, (4) sphincter impairment, (5) bedsore.

In myelomalacia, which is usually of small extent at first, girdle sensation is often absent or may be unilateral. Anesthesia at the onset is less complete; often unilateral. Paresthesia, numbness and tingling are often present without absolute anesthesia. "Dissociation" of the thermal sense or of heat or cold separately is often present on one side of the body or in one limb, and this side is the one *opposite to the motor impairment.*

In myelitis "girdle sense" is commonly present, numbness and tingling are followed rapidly by absolute bilateral and complete anesthesia for all forms of sensation below the level of the lesion; "dissociation" is not present as a rule.

In myelomalacia, *motor symptoms* are commonly *unilateral* at the beginning, affecting a limb or a muscle group. Muscular atrophy and R.D. correspond, and are often less extensive than the initial symptoms would lead us to expect, owing to re-establishment of circulation in part of the affected territory by contiguous vessels. In case of involvement of another vessel on the opposite side, the "level" often does not correspond as shown by the "pattern" of motor, sensory and trophic defects.

In myelitis, *paralysis* is commonly *bilateral, symmetrical*, and *complete* below the lesion in a few hours or days. Muscular atrophy and R.D. are bilateral and symmetrical in the territory supplied by the affected segments.

The *reflexes* in myelomalacia are commonly unlike on the two sides of the body. One knee-jerk may be gone, in lumbar lesions, the other simply diminished, sometimes accentuated.

The *plantar reflexes* are likewise apt to be *unlike*, e.g., a Babinski toe extension on the paralyzed side and a flexion or simply rigidity in the opposite foot.

In myelitis the knee-jerks are at first abolished or markedly diminished on *both sides*, later becoming accentuated if the mid-lumber segments be intact and the main lesion be not a "complete" one, which it practically seldom is. The *planter reflexes* at first as a rule are *also abolished;* a double "Babinski' toe extension appearing later.

Sphincter Control.—In myelomalacia, the *sphincters may escape* or may be affected for a few hours or days only.

In myelitis *complete prolonged loss* of sphincter control is the rule.

Bed-sores are often absent in myelomalacia and may be prevented entirely in most cases by care and cleanliness. The rarity of bedsores may possibly be due to the preponderance of the lesion in the anterior horns, circumscribed affections of which, as is well known, seldom or never cause bedsores.

In *myelitis*, on the contrary, *bedsores appear early* and are seldom preventable by any degree of care.

The absence of leucocytosis would be expected in myelomalacia; its presence in myelitis, as indicated by Starr.

The foregoing remarks on diagnosis may be tabulated as follows:

	MYELOMALACIA.		MYELITIS.
1.	No preceding disability generally.	1.	Preceding disability, injury or acute illness present.
2.	Onset of paralysis sudden.	2.	Onset of paralysis gradual or rapid; not sudden.
3.	Fever absent, pulse low-tension.	3.	Fever present, pulse high tension early in the disease.
4.	Chill absent.	4.	Chill sometimes present.
5.	Rigidity of spine absent, Spasm of extremities absent. Area affected variable often unilateral and slight in extent. Paralysis often monoplegic. Extension by sudden increments.	5.	Rigidity of spine may be present. Spasm of extremities often present. Area affected usually one or more entire segments. Paraylsis always paraplegic. Extension steadily progressive.
6.	Dissociation symptom (Thermo-anesthesia) often present on non-paralyzed limb or on both limbs in paraplegic cases.	6.	Dissociation symptom absent. Anesthesia bilateral and symmetrical to all sensations below lesion.
7.	Girdle symptom often absent.	7.	Girdle symptom present.
8.	Knee-jerks unlike, often unilaterally abolished.	8.	Knee-jerks abolished or diminished bilaterally and equally.
9.	Plantar reflexes unlike; unilateral "Babinski" sign common.	9.	Plantar reflexes abolished early on both sides (double "Babinski" sign later).
10.	Sphincteric control may remain of may be lost for a few days only.	10.	Sphincteric control abolished for a prolonged period.
11.	Bedsores often absent; usually so in small discrete lesions.	11.	Bedsores inevitable.
12.	Leucocytosis absent.	12.	Leucocytosis probably present.

THERAPEUTIC CONSIDERATIONS:

Early recognition and prompt treatment render the prognosis of myelomalacia much more favorable than that of myelitis. The main indications for treatment are obviously the same as they are in acute cerebral softening from thrombosis, namely:

1. To increase the blood pressure and encourage the peripheral circulation.

2. To promote liquefaction and limit organization in the inflammatory exudate which surrounds the softened area after a few days.

By meeting the first of these indications we improve the circulation not only in the territory adjacent to the blocked vessel, but likewise in vessels elsewhere, which may at that moment be undergoing a gradual occlusion. We hold the way open for the blood stream, by cardiac stimulants and arterial dilators, while our slower acting "alteratives" or "exudate destroyers," as mercury and the iodides should be called, are attacking the enemy already entrenched. By these measures much of the consecutive cicatricial pressure in the vicinity of the actual lesion is prevented and ascending and descending degenerations reduced to a minimum.

Rest on an air mattress with frequent changes of posture, extreme cleanliness, proper attention to the bladder and rectum, and after the acute stage, passive motion and massage of the paralyzed limbs; galvanism to the atrophied muscles and general hygienic measures complete the treatment. The results of such a plan of treatment must obviously average better than those of a merely "expectant plan."

The treatment of myelitis does not call so urgently for cardiac stimulants or arterial dilators; in fact the conditions may even furnish indications for opposite measures.

The spinal rigidity and spasms in the limbs, both of which are more apt to occur in myelitis than in myelomalacia, are best controlled by sulphonal in my experience, though bromides and chloral are also effective.

The "antiphlogistic" or "exudate destroying" effect of mercury and the iodides is not to be ignored in myelitis, even when syphilis is not present.

ILLUSTRATIVE CASES:

CASE I. Myelomalacia:

An active business man of 35, well-developed and of good habits, consulted me in reference to temporary loss of power in his legs a few days previous. There had been some premonitory numbness for a few hours and his feet felt "heavy." On his way home he had reached his premises when he suddenly sank to the ground, seemingly from weakness in both legs. There was no giddiness or mental confusion. He had not been ill nor drinking; nor had he experienced any injury or shock. He could not rise for some minutes, but did not call for assistance and finally managed to reach the house and retire to bed. He found it difficult to turn over while in bed, and on attempting to get up, he found he could only do so by rolling out and assisting himself with his hands. With some effort he managed to get about his business, feeling a sense of weakness in the legs.

After four days he consulted an oculist about some defect in vision and an exudative choroiditis was recognized. By reason of the history of weakness in legs he was referred to me for opinion and advice.

I found a healthy looking man, well-developed and nourished, who denied any knowledge of syphilis, used no alcohol, but smoked freely.

His gait was not visibly affected, but he still complained of an indefinite weakness in the legs. There was no "girdle" sensation. Pulse 108, very low tension, small volume. Sensation appeared normal to tact and pain everywhere, but an area of the outerborder of the left foot, extending from ankle to root of toes and nearly to the median line of the dorsal surface was insensitive to heat and cold.

The knee-jerks were unequal, the right about normal, the left decidedly diminished.

The plantar reflexes were also unlike, the right showing flexion and extension alternately, with occasional rigidity in a midway position, the left sharply flexor at all times.

A test for Oppenheim's reflex (by steady deep stroking at inner border of tibia) resulted in developing a distinct constant extension of the right great toe with flexion of the others. On the left foot the response was flexion of all toes.

The sphincters were not affected. Bedsore absent. Blood examination not made.

A diagnosis was made of thrombosis of a minute vessel, supplying portions of the IV and V lumbar segments of the right side.

The patient was put to bed, strychnia, nitroglycerin and strophanthus given to improve the circulation and mercury administered by hypodermic injection of "gray oil," alternating with iodopin given in the same manner.

The pulse improved, the sense of weight in legs and feet disappeared in a few days. In about two weeks the knee-jerks were alike and normal and the plantar reflexes were also normal and equal.

The patient has been well for eight months, with no further vascular accidents, and general good health.

CASE II. Myelomalacia:

A man of 40, colored, shop porter. Complete loss of power in right arm of sudden onset while sleeping. Three days later was admitted to my service at the Cincinnati Hospital.

Condition on Admission.—Man of average height, good general development. Habits good as to alcohol. Syphilis probable some years previous. Mental condition good. No speech defect.

Motion.—(See Fig. 1.)—Examination showed absolute flaccid paralysis of entire right upper extremity; even the scapula could not be moved by any voluntary effort. Is not conscious of any defect in left arm and uses it freely.

Tested by Matthieu dynamometer, however, the grasp in the left hand is only 55 *K* (estimated to be about 60 per cent. of normal). No facial weakness evident at this examination (see later). No complaint of leg weakness. Gait is good.

Sensation.—No subjective sense of pain, no dizziness, no mental confusion at onset or since. No loss of tactile or pain sense anywhere. Sense of heat and cold lost, or confused, in irregular area over the right shoulder anteriorly, over right pectoralis major and over outer half of right arm and forearm anteriorly to a point three inches above wrist. Cannot recognize cold with right hand or fingers. Cannot recognize heat or cold with left hand and over a longitudinal strip on posterior external surface of left arm and forearm. (See Fig. 2.)

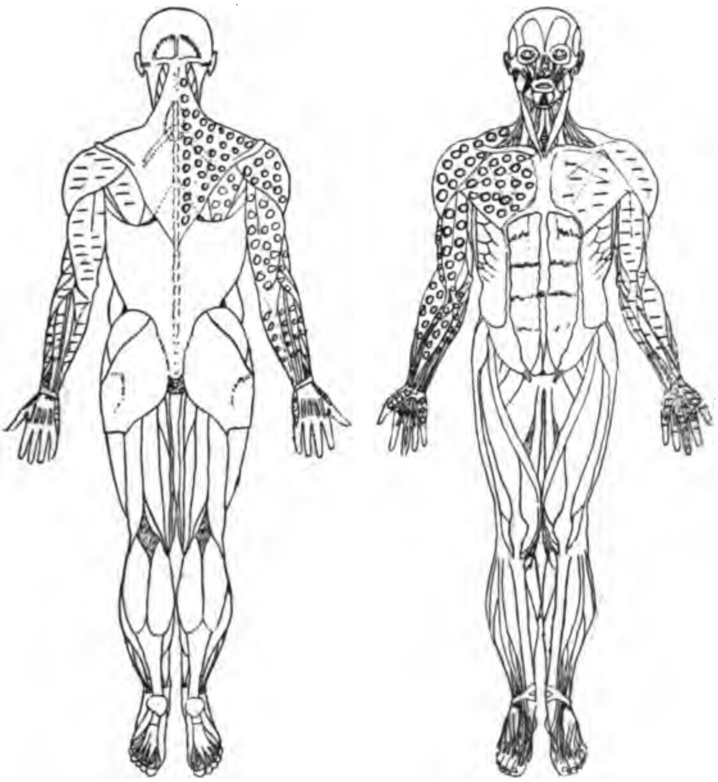

Fig. 1. Chart showing motor defects. Circles indicate absolute loss of power; dashes, partial loss.

Reflexes.—Pupils do not dilate in darkness or on irritation of skin of neck; they respond well to accommodation and convergence.

The supra-orbital reflexes are present and active right and left. Jaw-jerk absent. Elbow jerk present right and left. Wrist and supinator jerks present right and left. Hypochondriac reflex, right absent, left present clear and sharp. Inguinal reflexes present, right and left equal. Cremasteric reflexes absent. Knee-jerks absent right and left. Front tap contraction absent right and left. Tendo-Achilles jerks absent right and left. Plantar reflexes: Right, extension of great toe and flexion of all the smaller toes; a good "Babinski" response. Left, rigidity of great toe, with occasional slight extension; flexion smaller toes, incomplete "Babinski" response.

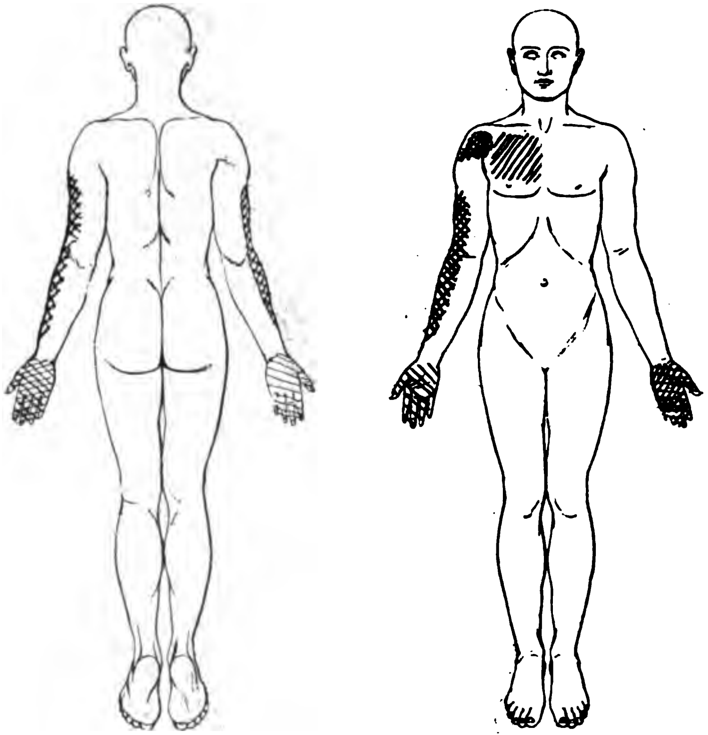

Fig. 2. Chart showing sensory defects.

Lines sloping from left to right—Loss of cold sense.
Lines sloping from right to left—Loss of heat sense.
Lines crossed—Loss of both heat and cold.

No sphincter defect. No trophic lesion. No obvious wasting. Pulse 96, low tension; temperature 98, respiration 16.

Seven days after the onset of the arm palsy (four days after admission to hospital) paralysis of right lower face and of the right orbicularis palpebrarum developed. The mouth was drawn to left, the tongue protruded to the right (actually, not apparently).

Electrical examination of the right arm and hand muscles at this date showed sluggish contraction to galvanism with polar reversal (A.C. > K.C.) in deltoid, and in thena and hypo-thenar groups, quick contraction with K.C. > A.C. in right trapezius, biceps, wrist flexors and extensors. Some wasting in arm was evident. The patient made a gradual improvement so that in two weeks he could flex the elbow slightly. One month later slight improvement manifest in flexion at elbow.

Five weeks later all movements of right arm and shoulder could be performed weakly, and patient could raise hand to top of head. Grasp of right hand registered 25 as a maximum, while the left registered 45%. (Reduction in power estimated at 75% in right grasp and 50% in left grasp.)

Unfortunately the patient left the house during my absence after a change of internes and the conditions of knee-jerks or pupils and other important items were not noted.

To sum up the conditions present in this case;—sudden onset, flaccid paralysis, absence of associated shoulder movements, the distribution of cutaneous sensory defects, the bi-lateral but unsymmetric character of the motor and sensory symptoms, the absence of mental confusion, or speech defect all point to a spinal location of the lesions. The partial recovery of function would indicate multiple minute areas of softening due to gummatous infiltration and thrombosis of numerous small vessels in the cervical cord from the III to VIII segments, rather than a single extensive lesion.

CASE III. Myelitis (primary):

As an example of true primary myelitis I cannot do better than refer to an abstract of the case reported at this meeting by my friends, Drs. Zenner and Wolfstein, through whose courtesy I had the privilege of studying it during life and afterward.

Man of 33, driver; general good health, gonorrhea within six months, followed by "la grippe." Syphilis not probable. Two weeks previous to coming under Dr. Zenner's observation his foot slipped in mounting his wagon step and he stepped forcibly to the ground. He went on with his work, but in two hours noted a numb feeling in his left side. The next day numbness in left leg and slight difficulty in walking. The right leg was affected similarly in a week. Then the right arm weakened and he was compelled to quit work. Three days later he lost control of the sphincters.

Three days after this Dr. Zenner saw him. He was completely paraplegic.

Temperature 101, pulse 108. Temperature during next twenty days varied between 100 and 102 usually, once rising to 104.

The paralysis extended and involved both arms and was preceded by spasmodic movements in right arm and hand. Dyspnea and dysphagia appeared. The knee-jerks disappeared; the right going first. The plantar reflexes were absent when patient was first seen. A "bedsore" developed over heel, four days after coming under observation (eighteen days after onset). Thirty-one days after onset, stupor, twitching of face muscles. Pulse 63, respiration 30. Death on the thirty-fourth day.

A diffused round-celled infiltration with no gross vascular lesion was found extending from the lumbar to the mid-cervical region. (See this JOURNAL, p. 190.)

As regards the relative frequency of myelomalacia and myelitis based on clinical observation, the following figures may be of some interest:

They are the results of a review of the neurological service in a large municipal hospital (The Cincinnati Hospital) for the past five years, lacking four months.

During this period 27,397 patients were treated in the hospital, of which 1,649 were admitted to the neurological service.

I have also tabulated separately the cases seen by me in private practice, and of which I have notes, during the same period.

Reviewing both sets of cases of "paraplegia" "myelitis," etc., during this period, I would tabulate them in accordance with present generally accepted views as follows:

				FINAL RESULT			
Hospital cases—	Number.	Males.	Females.	Practical Recovery.	Improved.	Died.	Not Stated.
Myelomalacia (syphilitic)	25	19	6	..	17	4	4
Myelomalacia (senile)	2	1	1	..	1	1	
Myelomalacia (typhoid)	2	1	1	..	..	1	1
Private cases—							
Myelomalacia (syphilitic)	14	13	1	4	8	2	
Myelomalacia (alcoholic?)	2	2	..	..	1	1	
Myelomalacia (la grippe)	1	1	..	..	1		
Myelomalacia (puerperal)	1	..	1	1			
Myelomalacia (senile)	1	..	1	..	1		
	48	37	11	5	29	9	5
Hospital cases—							
Myelitis (traumatic)	5	3	2	1	..	2	2
Myelitis (compression caries)	2	2	..	1	..	..	1
Myelitis (infectious)	4	3	1	..	..	1	3
Private cases—							
Myelitis (traumatic)	4	3	1	..	3	1	
Myelitis (gunshot)	3	2	1	..	1	2	
Myelitis (compression caries)	1	1	..	..	..	1	
Myelitis (sarcoma)	1	1	..	..	..	1	
Myelitis (hematorrhachis trau.)	1	1	..	1	..		
	21	16	5	3	4	8	6

TRAUMATIC EPILEPSY IN ITS MEDICO-LEGAL RELATIONS.*

By Arthur Conklin Brush, M.D.,
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Although the clinical phenomena of the epileptic condition have been recognized since very ancient times, yet when we come to the consideration of the medico-legal questions involved in the study of this subject, we again encounter the often repeated situation in medico-legal investigations; that the nature and limitations of the condition under consideration are as yet unsettled questions, for it clearly appears in evidence from the works of the recent writers on medicine that we are even at the present time unable to give an undisputed definition to the condition under consideration. Yet the aim of law is exactness: and as stated by Spratling "its medico-legal importance requires that the physician should be able to define what the malady is."

Strümpell defines it as "a frequent disease, characterized by paroxysmal losses of consciousness, and often associated with violent general convulsions."

Starr that "it is a disorder of the control of brain energy, due to weak or defective organization, and due to any lesion which weakens this control."

Gowers that epilepsy "is a disease in which there are convulsions of a certain type, or sudden losses or impairment of consciousness: but in which the convulsions are not due to active organic brain disease, reflex irritation or abnormal blood states."

Dana that "idiopathic epilepsy is a chronic functional disorder characterized by losses of consciousness and usually by convulsions: that mental disturbances may accompany or take the place of the convulsions: that symptomatic epilepsy is a form in which periodic convulsive attacks are due to gross organic brain changes: that Jacksonian epilepsy is a form of symptomatic epilepsy usually, is characterized by periodic convulsions affecting only certain groups of muscles, and often unattended by loss of consciousness: and that hystero-epilepsy is not epilepsy, but a form of hysteria."

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* Read before the New York Neurological Society, Dec. 6, 1904.

Oppenheim that "epilepsy is a disease which in its developed form is characterized by attacks of unconsciousness combined with convulsions."

Spratling that "epilepsy is a disease or disorder affecting the brain, characterized by recurrent paroxysms which are abrupt in appearance, variable in duration but generally short, and in which there is impairment or loss of consciousness, together with impairment or loss of motor coordination, with or without convulsions."

It appears then that the definitions of Starr, Dana, and Spratling are the only ones of these which can be made to include all the types of convulsions now grouped under the title of epilepsy: and as will appear later some of the types of traumatic forms would be excluded by such definitions as those of Strümpell and Oppenheim.

The same conflict of opinion also appears in the views of these writers as to the nature of the disease.

Strümpell states that of the actual causes we know nothing: that Brown-Séquard produced epilepsy in rabbits by injury to the cord, medulla, and peripheral nerves; that the progeny of these animals suffered from spontaneous epilepsy: that Westphal produced the disease in guinea-pigs by blows on the head and considered the disease due to minute hemorrhages in the medulla and cord: that the fact that between the paroxysms the patient shows no signs of disease, shows that there is no permanent macroscopical lesion: and that the cause is an intermittent functional irritation of the cortical cells.

Hamilton, that the fit is due to altered cell metabolism from toxic substances accumulating in the blood.

Gowers, that in most cases changes in the brain are not visible to the naked eye and are therefore termed idiopathic.

Oppenheim, that Chaslin found a gliosis of the cortex, which is confirmed by Bleuer.

Starr that epilepsy is usually if not always an organic disease, that there is no essential difference between the Jacksonian and the idiopathic forms: that the difference between them is in the extent of the spasm, and if in the Jacksonian form the convulsions become general consciousness is lost: that the organic nature of the disease is shown in its occurrence in mal-development of the brain: that it is the exception that epileptics are normal mentally and physically: that idiopathic epilepsy is due to gross and microscopical brain defects: that the influence of a bad heredity shows an inherited organically imperfect brain: and that many of the cases of so-called idiopathic epilepsy, especially those characterized by uniform motor or sensory aura, originate in acute encephalitis in infancy or childhood.

Spratling states that no satisfactory classification can be based on the etiology but that based on the symptomatology is: that Esheverra held that epilepsy is not a morbid entity and there is scarcely a disease of the nervous system in which epileptic convulsions might not happen: that Féré says that the convulsions experimentally produced in animals are not epileptic: that the fundamental factor in epilepsy is a loss of control over the inhibitory power of the cortical nerve cell's function to store up energy: that the lesions include almost every possible change of the cerebro-spinal axis; that for the most part these lesions occupy a secondary place in the causation: that as epilepsy is now regarded as a cortical disease only those lesions which involve the cortex are of pathological primary importance: that the toxic, dynamic, and gliosis theories are each inadequate: that the convulsions are due to a diseased state of the cortical sensory elements: that the most marked changes are found in the cells of the second cortical layer and especially in the nucleus: that the poison in epilepsy is a nuclear poison: that the neuroglia hyperplasia is due to the cell destruction and irritation of the toxins: that the epileptic impulse is transmitted by other than the ordinary motor paths: that heredity and trauma alone are insufficient causes: that there must be a toxin present but its nature is unknown: and that the disease is engrafted upon a cortical organic cellular anomaly induced largely by faulty heredity.

It appears then that the weight of evidence is in favor of epilepsy being considered as an organic disease of the cerebral cortex, which weakens the inhibitory power of the cortical cells: and it is further alleged that this degeneration and epileptic discharge only occur from the presence of certain unknown toxins. It would appear then if this latter theory is accepted that injury and disease of the cortex can produce epilepsy only in persons predisposed from the presence of this toxin. This theory can not as yet be accepted as proven until it can be shown that this toxin really exists, and that injury and disease are unable to produce the epileptiform state without its presence. Again, as it will afterwards appear, the assigned causes of epilepsy are very numerous and the number of epileptics very large, so it would appear as if this toxin must be present in a large percentage of human beings. The confusion which surrounds this subject has been increased by the use of the words "true epilepsy" by authors without their defining just what cases they would designate as such. At the present time it appears then that all our real knowledge consists in the fact that a certain type of convulsions can be originated from a great variety of causes, producing disease of the cerebral cortex.

The next question of importance is, Are the convulsions of which the plaintiff is alleged to suffer, of the epileptic type; and second, is there anything objective which would indicate whether they existed before the alleged injury? It is unfortunate that in these cases, we often have no guide beyond the history of the plaintiff, and difficult as it is to get a reliable history in private practice it is much more so in medico-legal work. This situation is often made worse by the confusion which exists in some medical minds between true and hystero-epilepsy. The accepted indications of a true epileptic seizure would seem to be its sudden onset, its occurrence during sleep, complete loss of consciousness in cases of general convulsions, the pallor followed by flushing or cyanosis of the face, the dilated and irresponsive pupils, loss of the corneal reflex, the turning of the eyes towards the side of a discharging lesion, the short sharp electric like nature of the spasms, the biting of the tongue in about half of the cases, the loss of the reflexes during several hours, the voiding of the urine and feces, the occurrence of petechiæ in the skin which do not fade for several days, a rise in temperature, a duration of only a few minutes, and a sequela of coma or headache. In answer to the second question, it is stated by Spratling "that in traumatic cases mental deterioration rarely occurs until after some years, and not then unless predisposition exists." It can not be disputed then that if only a year or two have elapsed since the injury and the patient shows well-marked dementia, and especially if this is combined with the well-known stigmata of degeneration, that the disease existed before the alleged trauma. The epileptic condition, as has been shown, has many assigned causes and the medical witness must therefore be able to exclude these before he can say with reasonable certainty that the alleged injury was the producing cause. From the study of these alleged causes, it again appears that there is a conflict of medical opinion as to the power of some of these assigned causes to produce epilepsy. Epilepsy is one of the most common diseases, occurring in one of every five hundred persons, and the larger number being idiopathic. The influence of a neurotic heredity seems to be unquestioned. It is stated to be present in one-third of all the cases, and a direct inheritance in one-third of these. The effect of chronic alcoholism in the parents is also undisputed. Chronic lead poisoning in the parents is assigned by Oppenheim and Spratling, and syphilis by Gowers, Dana, and Spratling. Spratling also assigns tuberculosis in the parents as a predisposing cause, and also rheumatism, scrofula, rickets, morphine, diabetes, chorea, and degenerations of the ovaries and testes; but Gowers denies that phthisis or gout in the parents has any relation. That idiopathic epilepsy is a disease of youth and young adult life seems to be undisputed. In the vast majority of the cases it develops before thirty, and in three-quarters of the cases before the age of twenty. Defective brain development as a cause also seems to be undisputed. Persons so predisposed frequently show some of the stigmata of degeneration. It appears then that predisposed persons can either develop epilepsy spontaneously, or from causes which would not be sufficient in persons not so predisposed; but it also appears that a congenitally healthy brain may become so diseased from such causes as alcohol, that any added source of irritation may result in an outbreak and thus the apparent cause may not be the real cause. This first view is supported by Starr, who states that "the influence of a bad heredity shows an inherited organically imperfect brain, and the supposed causes can only be accepted when they produce organic brain disease: by Gowers, who states that "the causes are usually inadequate except in cases with a powerful predisposition, and occurs as the result of a defective congenital brain development," and by Spratling "that in the majority of the cases both exciting and predisposing causes play a part."

The post-natal factors, outside of trauma, which are claimed as exciting or producing causes, can be divided into four classes: toxic, organic brain disease, mental, and reflex. Among the toxic causes the power of alcoholism to produce epilepsy appears to be unquestioned. Chronic lead poisoning is assigned by Oppenheim, but he is uncertain about cocaine, antipyrin, physostigmine, and chloroform. Spratling considers lead as a rare cause. The acute infectious diseases, especially scarlet fever, measles, and typhoid, are assigned as causes by all writers; and Starr believes that they do so by the formation of sclerotic plaques about the arteries. The power of gout is denied by Oppenheim and Gowers. Syphilis is considered as a common cause late in life by Oppenheim, Dana, and Spratling: but Strümpell considers that it has no direct connection with epilepsy. Starr and Gowers hold that syphilis produces epilepsy by causing disease of the vessels and membranes; but the latter considers it doubtful whether it can cause true epilepsy. Chronic nephritis is assigned as a cause by Oppenheim and Spratling, conditions of anemia and plethora by Strümpell, rickets by Dana, and Spratling holds that "derangements of the gastro-intestinal canal are of greater etiological importance than is generally supposed, that the gastro-intestinal cases are due to toxins either absorbed into the blood or act directly on the gastro-intestinal nerves: and that the influence of bad teeth and dentition is through the gastro-intestinal disorders so caused."

That organic diseases of the brain such as tumors, abscess, foci of sclerosis, hemorrhage, embolism, thrombosis, and arterial sclerosis are causes for convulsions of the epileptic type is undisputed: and after a time, it is stated by all writers, that these convulsions may resemble those of true epilepsy, but as already shown they do not define what the meaning of true epilepsy may be. Infantile encephalitis, meningitis, and post-natal palsies are as a rule undisputed causes, but Spratling holds that the polioencephalitis theory of Strümpell has received little corroboration.

Strong mental emotions, especially anger and terror, are undisputed exciting causes. Mental and physical over-work are also assigned by Strümpell and Spratling: but Starr considers the former as vague and uncertain.

The irritations which arise from scars or foreign bodies in any part are assigned as causes by Oppenheim, Golebiewski, Hamilton, and Dana: but Strümpell considers this etiological relationship as doubtful, Gowers as rare except in cases with a strong predisposition, Spratling considers it as possible but has never seen such a case, and Starr does not believe that epilepsy is ever so produced. Spratling adds that "the convulsions are epileptiform, like epilepsy, in the beginning": but as already shown he does not define what he means by true epilepsy.

Various diseases of the nose, larynx, uterus, intestinal worms, foreign bodies in the ears, ear diseases, errors of refraction, and carious teeth are assigned by Hamilton, Dana, Strümpell, and Spratling: but Gowers considers such causes as rare, and Oppenheim as not definitely shown. There seems to be no conflict of medical opinion that trauma is a competent cause for epilepsy, and according to Starr it is so in 11% of all the cases; but when the injury is so slight as not to produce fractures of the skull, severe cerebral contusion, or gross organic brain disease, it is a matter of grave doubt whether such trivial trauma can cause epilepsy in non-predisposed persons. Kirchhoff, Gowers, and Spratling state that cerebral concussion may produce a condition resembling true epilepsy: and this opinion is supported by Starr and Westphal who believe that in these cases, capillary hemorrhages occur, leaving a scar. Golebiewsky, Oppenheim, Bailey, and Hamilton concur in this view, but point out the fact that when epilepsy follows slight injuries there is usually a strong neurotic hereditary predisposition present, alcoholism, syphilis, or some nervous degeneration. Bailey holds that the general character of the convulsions would indicate that the disease was latent. Strümpell is somewhat conflicting in his statements, for he asserts that these cases may begin like genuine epilepsy, but are not genuine cases from the fact that there is an anatomical lesion and the symptoms may begin with a local spasm.

It appears then that in these cases assigned to slight cerebral contusions, in persons predisposed, where the symptoms were slight, where a considerable interval of time intervened between the injury and the onset of the epilepsy, and where the two were not connected by any mental or physical symptoms, that it can not be said that the disease would not have developed without the occurrence of the injury.

In cases with more serious injury to the head, as in fracture, especially depressed, or in injury to the brain, such as laceration, meningitis or hemorrhage, the evidence of the causal relationship of the trauma to the epilepsy seems to be undisputed: and the disease to be due to an irritation of the cortex from localized thickening of the cranium, splinters of bone invading the cortex, meningeal cicatrices, or localized inflammations. Van Gieson has shown that from these lesions the membranes become adherent, wedged-shaped areas of connective tissue extend into the brain, the cortical cells degenerate, and areas of gliosis form about the cortical arteries. Bailey, while admitting that these lesions form a pathological basis for the disease, denies that they explain the paroxysmal nature of the condition. Bailey also considers that the severity is the most important element of the injury, and that severe injuries are frequently followed by epilepsy.

Traumatic epilepsy may develop at once after the receipt of an injury, or not until after a period of months or years: but the largest number of cases develop within a year. Hamilton considers cases which develop at the end of two or three years as a rule suspicious, but admits that genuine cases have occurred after a period of five years. During this interval it must be admitted that the patient may appear normal, but more often gives a history of cerebral symptoms, such as mental weakness, irritability, headaches, or hemiplegia.

In the cases which are assigned to cerebral contusion the convulsions are from the first, as a rule, of the grand mal type: and in those cases due to coarse organic lesions, the convulsions may also from the first be of the grand mal type, but more often they begin as localized spasms without loss of consciousness. After a time the convulsions grow more and more extensive, and finally become general, and then consciousness is lost. Such paroxysms may be followed by a temporary loss of power in the muscles first convulsed, and if the lesion is progressive these muscles may become permanently paralyzed. From this study of these cases, it appears then: that for the medical witness to give an opinion with the reasonable certainty that the law requires, that the injury claimed was the cause of an epilepsy: that the plaintiff must be able to show that there were no evidences of the disease before the injury: the absence of other producing causes; that the disease occurred within a reasonable time after the injury: and that it was connected with the injury by some train of cerebral symptoms. For although it may be recognized in medicine that this connecting link may sometimes be absent, for the medical witness to claim that the disease is due to a thickening of the inner table or irritation of a splinter of bone, without any real knowledge that these conditions exist, is the purest speculation and has no place among the facts required in action at law.

Finally as to the future of these cases. This question, naturally from the conflict of opinion shown to exist concerning the previous questions, is also unsettled. So long then as we can not definitely say just which cases are to be included under the title of true epilepsy, opinions will differ as to the prognosis: and it is also evident that the general prognosis of the disease can not be in any individual case altogether admitted as satisfactory. It is evident then that our opinion as to the future must be influenced by the etiological class to which the plaintiff belongs and the individual surroundings which exist in his case.

As to the general prognosis of this condition: Oppenheim states that "epilepsy of itself does not shorten life," while Strümpell and Dana hold that it does to some extent: and Spratling points out that the condition of status which is often fatal occurs in one-third of the cases. Oppenheim states that recovery is rare, but more common than generally supposed, Dana states that from 5% to 10% recover, Spratling that 5% recover, and Strümpell points out that the natural history of this condition may cause it to disappear spontaneously and then return after many years. It is admitted by all writers that the prognosis is better in the grand mal type, in males, when there is a hereditary history, in the nocturnal form, when the convulsions are infrequent (three or more weeks apart), when mental changes have not occurred, when a considerable period separates the aura and the fit, and most authors hold, when it begins before the age of twenty, and Spratling considers that a beginning between 15 and 20 is of good prognosis. Long duration and mental weakness are as a rule regarded as of bad prognosis: but Spratling holds "epilepsy is not infrequently a curable disease, irrespective of its duration, but recent cases respond twice as quickly to treatment." Hamilton states that in cases due to organic cerebral lesions, surgical procedures have shown good results. This is not in accord with the observations of the writer, for in the five cases of his operated upon, though the convulsions disappeared for several months they afterwards returned. The removal of the cause in reflex cases according to Oppenheim and Hamilton has resulted in a cure: but Starr denies that their removal ever cured the disease.

To illustrate the points of this paper I have collected from my hospital records the histories of thirty-eight cases in which an injury was assigned as the producing cause. These can be divided into three classes.

The first class contains those cases in which, although put down on the hospital records as traumatic, careful analyses reveals the fact that the assigned cause can not be said with any reasonable certainty to be the real producing cause.

There were ten cases of this class. In eight the injury was trivial and the immediate symptoms produced were also in seven. The ages at which the epilepsy developed in eight of the cases was from five to twenty years, the usual period for the appearance of the idiopathic form. The interval which elapsed between the injury and the epilepsy varied from two to twelve years. During this interval there was no evidence of any cerebral trouble. Three of the patients gave a family history of epilepsy in the parents, two of insanity, one of a discharge from the ear, and in this the spasms were at first confined to the opposite side of the body, and of the two cases which were over twenty years of age one suffered from alcoholism and the other from syphilis. In all the convulsions were of the grand mal type. In the second class are grouped those cases in which the injury to the head produced a well-marked state of cerebral contusion, which seemed to be the only recognizable cause for the epilepsy. In all the injury to the head was severe, producing marked cerebral symptoms lasting from four hours to three days.

In six cases the first convulsion occurred within twenty-four hours after the symptoms of cerebral contusion subsided: and in the remaining four the interval varied from three days to four months. During this period the patients suffered from headache, vertigo, and mental confusion. All but three of the patients were over twenty-five years of age. The convulsions were all of the grand mal type. In none was there any evidence of other causes.

The third class contains those cases in which there were evidences of organic cerebral injury. Of these there were sixteen cases. Their ages varied from twelve to forty-four years. In all the injury to the head was very severe. In all the coma lasted from nine to twenty-four hours. In eight there were depressed fractures of the vault, and in three, of the base. Two had paralysis of an arm, and eight hemiplegia. In twelve the convulsions were at first of the Jacksonian type, gradually passing into grand mal, and in four of the latter type from the first. These four developed immediately, and in the others the length of time before the convulsions appeared varied from three days to four years.

It will be seen then that in this last group of cases there can be no question that there was a brain injury, and that it acted as the cause: and there was nothing in the histories of these patients to suggest the existence of any other etiological factor. If such injuries to the brain can produce epilepsy the trauma must be accepted as the sole cause: but if the theory that injury can only do so in persons predisposed by the presence of an unknown toxin in their blood, then injury can only be accepted as the exciting cause. It appears then that from a medico-legal standpoint our knowledge as yet will only enable us to state that certain injuries to the brain can excite epilepsy: and that the toxic theories can not be advanced as facts until there is some evidence upon which to base such statements: and if this latter statement be true it must be shown that this toxemia is present before the physician can say that the disease was the immediate and necessary result of the injury.

Society Proceedings

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

November 17, 1904.

Dr. P. C. Knapp in the Chair.

A Case of Brain Tumor.——This was reported by Dr. Walton. The case was of interest through its atypical course, and the presence of symptoms suggesting local encephalitis.

Mrs. G., aged 63, seen in consultation with Drs. Griffin and Ryder, of nervous disposition and inclined to worry, but with no prior mental disease, appeared well up to June, 1904, though is said to have talked and acted strangely at times for some months before, apparently sometimes appearing not quite oriented. There was no history of grippe or other known infection, no abdominal, pulmonary, cardiac or renal complication. Prior to the attack in June she had had considerable trouble and worry over household affairs. She became restless, would roam about and talk at random; complained of severe headache. Is said also to have been aphasic. After about a week, went to bed semi-comatose. From this time the temperature was elevated, twice reaching 103.5 F. with chill. Catheterization was necessary. Some improvement in mental condition followed. Mental torpor ensued and tenderness on turning head; no retraction. About September 1st hemiplegia appeared. Examination one week later showed optic neuritis. Blood count: whites, 12,500.

When Dr. Walton saw her September 19th she lay quietly on her back without appearance of discomfort. The pupils were alike and reacted to light. On being addressed, she would look quietly at the speaker without responding. On being urged to answer, would sometimes make an irrelevant reply; for example, when asked what she had in her hand, finally said, "Your voice is very much like Mrs. X.'s." Did not recognize objects in right hand, evidently recognized them in the left. Apparent loss of temperature sense in right arm and leg. Movements of right arm and leg restricted; left, normal. Could not name handkerchief, but says it is something to wipe nose with. Did not protrude tongue or touch nose with finger upon order, but could imitate these movements. Knee-jerks absent on both sides. No clonus. Tendency to Babinski on the *left.*

Condition remained practically the same except with deepening torpor and more marked hemiplegia, but without sign of headache, until a few days before her death, which occurred Oct. 15th.

Dr. McGrath, who performed the autopsy, reports a glioma in the following situation: "The mass extends anteriorly to within 2.5 cm. of the surface of the frontal lobe; inwards to the median line; outwards nearly to the surface of the frontal lobe, just anterior to the fissure of Sylvius, the cortex at this point showing some softening; downwards to within about 1 cm. of the surface of the base; and backwards to a point which externally is 2.5 cm. posterior to the tip of the temporal lobe, and internally corresponds with the front of the optic thalamus. This mass occupies the greater part of the corpus striatum, replacing the body of the caudate nucleus, the most of the lenticular nucleus, and the anterior portion of the internal capsule. Its peripheral parts infiltrate the white matter contiguous, and externally the gray, within the limits above specified."

Dr. Knapp said that the history given him differed somewhat from that obtained by Dr. Walton. When Dr. Knapp saw her in the middle of September he was told that she had been dyspeptic in the spring, with some cardiac weakness, that she had been much quieter than usual, and that her memory had failed. She had had considerable headache, but not severe in character, so that her attention could readily be diverted from it. In June she could not remember words, often used the wrong word, and then realized her mistake. Her eyes looked wild and unnatural. About the first of July she went to bed, was unable to help herself, passed urine and feces in bed, but moaned when wet. There was no somnolence. For ten days she had helped herself a little. Her husband said that she understood all that he said to her and answered him properly. Her attendant physician stated that she rarely answered him at all, or else made some irrelevant remark. While Dr. Knapp was there she answered no question, executed no command, and paid no attention to a written command. She uttered one sentence, well articulated and properly arranged, relating to the general situation, but having no relevance to what was said or done at the time. The blood count showed 6,800,000 reds and 12,500 whites. There had been occasional chills and elevations of temperature. There was tenderness and rigidity of the muscles on the left side of the neck. She had old catarrhal otitis with labyrinthine trouble, but no source for a cerebral abscess could be elicited. There was well-marked right hemianesthesia without any hemiplegia. This had existed for some weeks. Dr. Jones reported optic neuritis, greater in the left eye. The pupils and visual fields were apparently normal. The reflexes were lost. There was no vertigo or vomiting. The chills and tenderness of the neck suggested abscess, but as no source could be found, the diagnosis of tumor seemed most probable. Dr. Knapp believed the tumor to be deeply seated, involving the insula and thalamus, but it proved farther forward that he had thought.

A Case of Tumor of the Frontal Lobe.—This was reported by Dr. Brownrigg, illustrating anew the rather remarkable absence of crippling symptoms during the prolonged growth and softening of a large tumor mass in the frontal lobe. A man of 51 became semi-comatose from the effects of intracranial pressure. Severe headaches recurred periodically for over five years, but had never incapacitated him for active work. He had been in the habit of taking morphine and headache powders for his head pains, and was thought by his physician to be suffering from the withdrawal of the former drug when first seen. He could be roused and would talk connectedly and enunciate well, though he was apathetic and tended to sleep. He still complained of generalized headaches. His superficial reflexes were abolished, knee-jerks diminished, and ankle clonus absent. He was removed to the Highland Spring Sanatorium, and gradually became continuously comatose and died, after having been acutely sick and prostrated only two weeks. Towards the end the muscles of the left side, especially of the arm and leg, became more spastic than those of the other side; the left knee-jerk increased, and the increase of the plantar reflex became especially noticeable. Throughout his semi-comatose condition he frequently put his right hand over the right frontal region, though he would never say that the pain was greater there. The diagnosis of tumor of the right frontal lobe region was confirmed at autopsy. The brain showed great increase in size externally to correspond to the cavity apparent to all, approximately the size of a hen's egg. The cavity was filled with soft degenerated brain tissue and partly absorbed blood clot, and was probably due mostly to vascular changes and slow softening. There was no history of shock. There was reason to believe that syphilis existed and was partially treated ten years ago. Without much doubt, this man had been carrying this mass of dead tissue for months, and it is especially remarkable that he was able to be about to so late a date. In fact, he drove trotting horses for exercise each day up to two weeks before death with apparently perfect control of himself and the animals.

Dr. Walton said that these tumors add to the numerous instances indicating that diminution may result from cerebral disease. The fact that in the second case the reflexes returned and became exaggerated only tends to show that the spinal reflex centers were not the original sufferers and were able to take on their function after an interval, as happens in certain cases of cerebral hemorrhage and cerebral trauma in which the reflexes disappear for a number of days, but return and become overactive. It is true that in slowly progressive cerebral lesion, exaggeration rather than diminution of reflex is the rule, but the first case reported tends to show that cerebral lesion alone may cause even persistent loss. The best explanation of the various phenomena seems to be the assumption in the human mechanism of multiple centers for the reflexes, of which the cerebral are those of primary control.

Dr. Knapp said that as the cord was not examined in either of these cases, it seems hardly fair to cite them in support of any theory as to the location of the reflexes. Absence of the reflexes is not very common in cases of tumor, while changes in the cord are often found, which may be enough to explain such absence.

A Case of Cholesterine Stones in the Brain of a Man of Fifty-six, Dead of Heart Disease.—This case was reported by Dr. E. E. Southard. The brain showed extensive arteriosclerosis, with microscopic signs in the secondary distributing system. There were infarctions of lung and kidney, besides a small fresh area of softening in the cerebrum. The largest mass of cholesterin crystals weighed 2.25 grams, and measured 2 cm. in diameter. The mass occupied the middle of the left lenticular nucleus, and was surrounded by a thin neuroglia capsule. Two smaller masses lay in the cortical gray matter.

Both gray and white matter of the spinal cord at various levels showed miliary gliosis about masses of cholesterin crystals. The patient had been under observation three weeks and had shown no nerve symptoms. A frost-bite on the right foot had failed to heal in a period of some months.

A Medico-Legal Study of Some Noted Criminal Cases.—This was read by Dr. Folsom.

Dr. Stedman was glad that the reader had brought to light the old case of Marie Jeanneret, because of its resemblance to that of Jane Toppan. Both were nurses, and both poisoned without adequate motive a large number of patients. Dr. Stedman had been inclined to make use of this parallelism by coupling the two cases in his account of the mental condition of Jane Toppan, but selected instead that of Christiana Edmonds, which was better reported and in important respects more striking in its similarity. There is little doubt that, with the light that has been thrown on this class of cases in the last half century or so, and since Marie Jeanneret flourished, she would have been held to be, as were the Edmonds and Toppan poisoners, insane and irresponsible. In fact, Lombroso, who, when there is a question between criminality and insanity in a given case, is especially prone to consider the person simply a criminal, classes Marie Jeanneret (in an abstract of her case) among the criminal insane of the hysterical type. Jesse Pomeroy is, in Dr. Stedman's opinion, a typical and extreme case of normal insanity, utterly irresponsible and unpunishable, and would be so classified nowadays by most authorities on a full examination of his history and mental condition.

Insane murderers of this degenerate class do not, in his opinion, deserve the extreme penalty of execution or imprisonment for life, nor would such a penalty have any deterrant effect on other lunatics. It would also tend to revive the old retaliative idea of punishment, and was neither good law nor good medicine. He regretted the advocacy of such a measure by Dr. Folson, as he believed it to be retrograde in its tendency.

Life confinement in an asylum for the criminal insane, on the other hand, meets all the requirements in the way of justice, humanity, medical care, and, if you will, punishment.

He was inclined to agree with Prof. Hyslop of Columbia that the only way to make any headway against murders by anarchists was to instill into the minds of the public the fact that anarchism is a stamp of insanity, and that public and continual expression of anarchistic sentiments should be the signal for the commitment to the insane asylum of those who are guilty of it, just as we endeavor to protect society from the delusionally insane when they show signs of endangering the lives of others.

Mr. Koren said that his interest in the subject under discussion extends beyond capital cases; and he wished. therefore, to make a plea for the wider application of the science for which this society stands in the treatment of criminals.

The whole modern penal system rests upon the assumption that the criminal is, generally speaking, a normal being. The criminal codes, therefore, virtually declare the insane criminals, because not normal, to be outside of the pale of the law, and do not permit their punishment. In other words, the sole distinction made is between responsible and irresponsible criminals. To him this classification seems not only unscientific, but one leading to the most deplorable results in practice. Clearly, there is between the two groups distinguished in the law a third, consisting of persons who lack the ordinary moral perceptions and are thus only partially responsible, without it being possible to declare them insane in the usual meaning of the term.

It does not make any difference how one views the doctrine of "moral insanity," or how one theorizes about the "born criminal" as an anthropological variety of the human species, the fact remains—and we have to thank Lombroso for emphasizing the need of dealing practically with the matter—that many men and women again and again come into conflict with the law because they in some respects are abnormal. To put it differently, there are in our penitentiaries and houses of correction large percentages of incorrigible habitual offenders who have become such chiefly because their individuality deviates from the normal type. He supposed the explanation lies in some congenital or acquired abnormal condition of the brain.

How are we dealing with the members of this group? As a rule, in the application of penalties the law makes no distinction between them and the normal individuals. In exceptional cases in which indubitable evidence of insanity exists, they are declared irresponsible and treated as sick persons, not as criminals. Most of them are sentenced according to a more or less fanciful scale of penalties which presumes to fit the crime, but pays little or no heed to the mental traits and peculiarities which have tended to make the individual in question a criminal.

The result is that we fail both in just dealing with many criminals and in affording society adequate protection against them.

The point he wished to make is that our criminal codes must bear the opprobrium of being antiquated, unscientific, and even barbarous, until they distinguish between three groups of criminals: First. those who are wholly responsible for their acts; second, those who are only partially responsible; and third, those who are wholly irresponsible, that is, insane.

Self-evidently, no innovations are necessary for dealing with the first and third groups. The second he would distribute according to the needs of individuals among institutions other than those of a penal character; for instance, the epileptics, certain alcoholics, the feeble-minded, etc. For a number of incorrigible habitual offenders it might be necessary to establish separate institutions in order to secure their permanent segregation.

He was not dismayed by the thought that he had hinted at measures which may seem to some too ideal, and therefore impossible in practice.

We have begun to move out of the old ruts. There is a growing movement for a more rational treatment of criminals, based on a scientific classification in the courts. It remains for a body like this to give this movement a safe direction.

Dr. Copp said that the practical side of this matter relates to the public safety. There is no doubt that such persons are abnormal, being either mentally deficient or mentally diseased. The question is one of degree of responsibility. Should such persons be dealt with as responsible or irresponsible persons? The right principle should be applicable in dealing with lesser as well as capital offenders.

The present penalty for a capital offense is death or confinement in an insane asylum for life. Therefore, the proposed change to life imprisonment would not affect the public safety in this class of cases. It would allow the doubtful case of insanity to be confined in a prison for life; in other words, it might substitute prison methods for hospital methods. But in dealing with minor offenses the principle of responsibility affords distinctly less protection than that of irresponsibility. If a mentally abnormal person is regarded as responsible he is arrested, imprisoned for a short time and discharged into society. The process is repeated indefinitely, perhaps until he has committed a capital offense. On the other hand, if he were regarded as irresponsible he would be committed at once to an insane hospital or the school for the feeble-minded, and would be permanently secluded from the community. Public safety would be efficiently guarded, and the commission of a capital crime might be prevented.

Dr. Mitchell thought it was desirable to give all men accused of crime the benefit of any doubt that might arise as to their responsibility, on account of mental defect or disease.

The question of responsibility was usually carefully determined in the more serious offences, but he knew of many cases where men had been convicted of minor offenses and misdemeanors committed subsequently to the onset of a psychosis.

This was a hardship that rested fully as heavily upon the family of the accused as upon the individual.

He felt that society should be carefully protected from the possibility of further offenses in the case of persons acquitted of serious crimes against the person, on the ground of irresponsibility, due to chronic or congenital causes, and that such persons should be held under order of the courts, their liberty being granted only after most careful consideration of the individual case and the nature of the offense.

He spoke of cases where this caution had seemingly been disregarded.

Dr. Knapp saw no necessity for any change in the present law. The jury now have it in their power to accomplish the result aimed at by bringing in a verdict of murder in the first or second degree, a privilege which they did not have under the United States law in the Bram trial. If the jury prove too severe, there is plenty of opportunity for the judge or the governor to alter the verdict. The tendency to insist upon the complete responsibility of insane and defectives was hardly to be commended, and adherence to judicial decisions as to the prisoner's ability to distinguish between right and wrong or to the older legal opinions had only too often led to the judicial murder of the insane and irresponsible, as in the Guiteau case. The whole doctrine of human responsibility, and even the freedom of the human will, were to-day in dispute. No one who had to do with the criminal classes could doubt that many of them were at least defective and with limited responsibility, and it was improper to judge them by the standards of the normal man. Some day it might be possible to make the diagnosis of criminality before the subject had committed any overt act and put him under restraint, as we do the homicidal paranoiac.

It might be well to get rid of the term "moral insanity," but it would be better, if we could, by abolishing the term, to get rid of the morally insane.

CHICAGO NEUROLOGICAL SOCIETY.

November 17, 1904

The President, DR. SYDNEY KUH, in the Chair.

Probable Multiple Sclerosis, with Suspicious Family History.—This case was presented by Dr. L. Harrison Mettler. The patient was 32 years of age, married ten years, and had had two children, both well. The wife had been perfectly well. There was no neuropathic history, except as follows: The paternal grandparents were first cousins; the paternal uncle, aunt and the patient's father were congenital deaf mutes; the patient's mother was rendered a deaf mute in childhood from scarlet fever, and he himself was the weaker of twins. He had the childhood diseases without sequelæ, and denied specific infection, living a moderate life, and was perfectly well up to the time of his marriage.

Eight years ago he first noticed the beginning of his trouble, when he was taken with a relatively sudden numbness in one hand, and later on, in the leg of the same side. He dragged his numb foot for quite a time. Patient subsequently had two attacks of numbness, paresis, weakness and dragging of his foot, and his condition grew worse each time. His wife described his speech as difficult and delayed, "as though he had his mouth full, and jerky." Dr. Mettler said he did not show much of this difficulty. No headache, no pains, no sensory symptoms, no bladder trouble. A year ago last August he had a third similar attack. The patient's eyesight had been unaffected until recently. There was some slight paleness of the disk in July. The mental condition was stated to be of the markedly depressed, melancholic type.

Dr. Chas. Mix said that the patient showed a paralysis of the left internal rectus, a little adduction of the foot, hardly any paresis, but much spasticity, and that his extensors were stronger than his flexors. The right pupil was suspicious. These irregularities were important points in establishing a diagnosis. Dr. Mix added that of the four cardinal symptoms of disseminated sclerosis three were absent, and asked if there were any possibility of optic neuritis or optic atrophy.

Dr. Chas. Lodor raised the question of the condition of the vascular system, and said that the heart skipped every third or fourth beat, that there was a murmur heard at the apex of the heart, and a decided aortic click. The radial artery on the right side was certainly very much harder than is usual at 32 years, and that there was a marked sclerosis, such as is seen in the secondary stages of syphilitic troubles.

Dr. Harold N. Moyer thought the diagnosis of multiple sclerosis presented the highest possibility, though the case was not typical. He further said that the disseminated scleroses do present some of the most extraordinary pictures at times, and a great many things pointed to that diagnosis in the case presented. One point was the mental and emotional state. Nothing clearly excluded multiple sclerosis. That he had combined degeneration the history, the progress and the clinical picture made evident, and for reasons stated, Dr. Moyer believed multiple sclerosis was the far more probable lesion. Its distribution in any given case determined the symptoms largely, and diagnosis was often not possible for years after the commencement of the disease.

Dr. Sydney Kuh said that it seemed to him that the diagnosis lay between two things: either a disseminated sclerosis or else syphilis of the central nervous system, and while all the symptoms might occur in both, they would be more likely in syphilis than in disseminated sclerosis. The condition of the pupils would be more likely to occur in sclerosis. This patient had an irregular pupil. The early occurrence of sensory disturbance, the early appearance of bladder symptoms, the contrast between the very slight rigidity and the spastic gait suggested the possibility of syphilis. There could be no possible harm in gentle, moderate antisyphilitic treatment, and good might come of it.

Dr. Specht said that this had been done six years ago, and carried on for months. The patient had had potassium iodide, 10-15 grains, three times a day for months, followed by the iodide of mercury, a considerable dose, for some time. He seemed to get better at times, but the gain apparently could not be attributed to the remedies.

Dr. Mettler brought up the question as to whether any reliance might be placed upon the family history, and whether it might be a factor in producing the patient's present condition. It seemed so to him. There was a degenerative condition running through the family, or at least there were defectives. Dr. Mettler did not think the man had syphilis, and closed the discussion by saying that he was especially interested in the pathological side. It had been laid down that multiple sclerosis is absolutely nonhereditary, yet a few cases have been reported where mother and daughter had multiple sclerosis, and a number have been reported where heredity played a striking rôle, bringing the disease in the class of degenerative troubles, and some have thought that there were two conditions, and one of these a family type. Five cases were reported in one family. Taking everything into consideration, Dr. Mettler thought that if the diagnosis be correct, there may be something in heredity that might change the hard-and-fast rule enunciated.

Progressive Bulbar Palsy, with Beginning Amyotrophic Lateral Sclerosis.—This case was presented by Dr. Julius Grinker, who said that the patient was 58 years old and had not been able to speak for some time, though his intelligence was good. Some time after the dysarthria had appeared the man had experienced difficulty in swallowing, particularly liquids. This condition had grown worse, so that life was maintained by ingesting small quantities of food at short intervals. The patient had frequently to assist the bolus of food with the finger or spoon towards or into the pharynx. Choking spells were not unusual with him. Of late he complained that his head had become so weak that he was compelled to hold it up with his right hand to keep it from falling forward.

The patient was an old man of medium height, with head resting upon his sternum and chin somewhat inclined toward the right. Saliva constantly dribbled from his mouth and could be seen in streaks upon his clothing. The mouth appeared as a large transverse fissure, devoid of expression, and in marked contrast with the upper portion of the face, which showed numerous wrinkles. The tongue lay passively on the floor of the mouth and could not be protruded. It was corrugated, somewhat atrophied and of a peculiar velvety feel. He was not able with any amount of exertion to touch his cheek with his tongue, and could barely lift it from the floor and place it above his lower teeth. For practical purposes, the tongue was motionless. The lips were rather thin and flabby and stood wide apart. A request to pout his lips as in kissing or whistling elicited no response. Upon requesting him to say "Ah," the pillars of the fauces seemed inactive, but upon tickling his uvula there was some response, and a reflex obtained on tickling his fauces. A laryngoscopic examination discovered the vocal cords in the cadaveric position. The muscles of mastication appeared weak, the right sterno-mastoid atrophied, and the left had almost entirely disappeared. The extensors sustained the entire weight of the head. The special senses were uninvolved, and there was no sensory disturbance of any kind. The deep reflexes of the upper and lower extremities were markedly exaggerated. Upon tapping the chin a jaw clonus was elicited, which was better felt than seen. There was well-marked generalized atrophy of the upper extremities. The shoulder muscles were weak, and there was a cervico-dorsal kyphosis. Unaided, the patient could not succeed in undressing himself. The thenar eminence of the left hand was markedly flattened, as well as the palm of that hand, while the right hand showed a similar atrophy in the hypothenar eminence. There was an extensive scar on the left hand, the result of an injury received several years ago, which he claimed to be responsible for the weakness of that hand. A dynamometer registered 60 in the left hand and but 20 in the right. Besides the sterno-mastoid muscles, the trapezii were atrophied in the upper portion; the deltoids, the biceps and triceps, the left supraspinatus, the right infraspinatus and the right pectoral were all more or less atrophied. The gait was about normal, but the left foot occasionally scraped the floor. Some stiffness in the left ankle joint was said to be the result of rheumatism, which would probably account for this scraping. The Babinski phenomenon could not be obtained, and no appreciable diminution of strength could be detected in the lower extremities. Fibrillar tremor in shoulder and arm muscles could occasionally be seen. There was no reaction of degeneration, but a decided quantitative reduction of both currents in the tongue, lips and mastoid muscles. The pulse was 90 to the minute.

To summarize, Dr. Grinker said this was a slowly progressive bilateral affection of the motor nuclei in the medulla, involving principally the hypoglossal, spinal accessory glossopharyngeal, lower facial and probably some portion of the trigeminus and vagus. He said that an interesting question arises as to whether this be the disease described by Duchenne as glosso-labio-laryngeal paralysis, commonly called progressive bulbar palsy, as a distinct entity; or, was the symptom-complex, progressive bulbar palsy, but the beginning or end of some other disease? Progressive muscular atrophy, pseudo-bulbar palsy, apoplectic bulbar palsy and amyotrophic lateral sclerosis were to be considered. Against progressive muscular atrophy would be the presence of exaggerated reflexes everywhere, and the absence of the reaction of degeneration in the paretic muscles. Apoplectic bulbar palsy has a sudden onset, and inclines toward a hemiplegic distribution. Pseudo-bulbar palsy might produce exaggerated reflexes and a picture resembling the case presented, but there was no history of two different attacks, and the bulbar palsy was complete, while in the pseudo-bulbar variety the palsy is never complete, and some disturbance of the intelligence is the rule. Pontine tumor could also be excluded on account of the absence of tumor symptoms. In amyotrophic lateral sclerosis there is involvement of both the central and peripheral neurones, either in the cord or in the medulla oblongata, or in both. Though it usually begins in the cervical cord and later extends upward, it may begin in the medulla oblongata and extend downward.

In the case presented there was masseter clonus, exaggerated reflexes, and weakness of muscles in the upper extremities and shoulder girdle, the paretic muscles reacting to both currents fairly well. This would indicate that the central portion of the nuclear representation in the medulla must be involved in addition to the nuclei themselves, as well as the pyramidal tracts and their continuation in the anterior cornual cells. The diagnosis of amyotrophic lateral sclerosis with a bulbar beginning appealed to Dr. Grinker as the most logical, although the amyotrophic lateral sclerosis was still in the developing stage.

Multiple Cerebral Gummata.—Dr. James B. Robb showed sections of a brain containing multiple gummata and reported the history of a case, saying that the patient, a laborer, had begun to show symptoms three weeks before death, and that his condition was attributed to a fall which had produced a slight scalp wound. Following the injury, the patient had complained of headache, talked irrationally, was unable to eat or sleep, was suspicious, imagined he was pursued, saw objects on the walls, talked to himself, walked unsteadily and vomited at times. The doctor said no history of previous illness nor of venereal infection was obtained.

Examination showed a heavily-built, well developed young man, who walked to the hospital, but with an uncertain, staggering gait; excited and irrational, so that restraint was necessary. The temperature was subnormal, the pulse slow, 58, irregular in time and volume. Respirations were stertorous and irregular, approaching the Cheyne-Stokes type. The heart and lungs were normal. The abdomen showed no abnormalities, the liver and spleen not being palpable. There was a scar on the left side of the glans penis. No spasms or paralyses were observed in face or extremities. The cremasteric and plantar reflexes were present. The patellar reflexes were not obtained, probably on account of the difficulty arising from the patient being in constant motion. The posterior cervical and epitrochlear glands were not palpable. No deviations nor nystagmus was observed in the eyes, but the right pupil was larger than the left. Examination of the fundus showed bilateral choked disk of extreme degree.

The patient sank into coma and died in less than 24 hours after entering the hospital. Permission to examine the chest and abdomen postmortem could not be obtained, but the brain was removed and revealed the following:

Marked congestion of all the vessels of the meninges; moderate dilatation of the ventricles with a clear fluid. On the mesial surface of the left frontal lobe, 3 c.m. behind the frontal pole and 2½ c.m. below the upper, was a nodule measuring 1½ c.m. along the mesial surface and 7 m.m. transversely. It was entirely in front of the corpus callosum, firm in consistency, of the same color as the white matter of the brain, apparently covered by pia, and everywhere displacing the cortex and separated from it. Eight c.m. in front of the left occipital pole on the inferior aspect of the lateral surface was another globular subpial nodule, 1 c.m. in diameter, answering in all respects to a description of the first. Seven and a half c.m. in front of the occipital pole on section on its anterior aspect, 5 m.m. below the external surface, was a firm, grayish area, which extended 17 m.m. inwards and measured 13 m.m. from above downward. It was directly connected with the brain substance, but quite sharply circumscribed, and interrupted a sulcus which extended 5 m.m. inward from the inner portion of the nodule. The bottom of this sulcus was located 2 c.m. from the posterior horn of the lateral ventricle. This section was a vertical one made 7 m.m. behind the opening of the aqueduct of Sylvius into the fourth ventricle. Just below and external to this was a firm, globular supial nodule 7 m.m. in diameter, resembling the first two above described. Sections before and behind that just described showed the greatest porterior measurement of the largest tumor to be not more than 15 m.m. One section 7 c.m. behind the frontal pole, going through the anterior commisure, extending from the bottom of the Sylvian fissure upward and outward as far as the external capsule, showed a dark, grayish-white area, quite sharply outlined and soft, measuring 17 m.m. in length and 4 m.m. in breadth, bordered above by the lenticular nucleus and below by the gray and white matter of the temporal lobe. Nothing was seen of this area 5 m.m. in front of or behind the section described.

Microscopic examination of the tumors showed typical syphilitic gummata with endarteritis and periarteritis of all the meningeal vessels adjacent to them. Several sections taken from various parts of the brain where no gross lesion existed showed the presence of a universal endarteritis and periarteritis, both in the meninges and cerebral cortex.

The case was of interest, first, on account of the classical symptom-complex of brain tumor, no one symptom except focal manifestations being absent; second, the pronounced character of the symptoms and the rapid course as contrasted with the insignificant gross lesions, which might have conceivably been overlooked had the history been absent at post-mortem; third, the fact that the man died of a curable disease, the diagnosis made by the physician in charge having been delirium tremens.

Periscope

REVUE DE PSYCHIATRIE ET DE PSYCHOLOGIE EXPERIMENTALE

(October, 1904.)

1. The Researches of M. Mourly Vold on the Visual Hallucinations of Dreams and of the Waking State. VASCHIDE.
2. Contribution to the History of Degeneracy. The Myopsychics of Joffroy, or Association of Muscular and Psychic Troubles. VURPAS.
3. Pathological Anatomy and Nature of Dementia Præcox. KLIPPEL.
4. Sugar Superalimentation. DAMAYE.

1. *Visual Hallucinations.*—This article is a review of the work done by Prof. Vold of the University of Christiana. The principal conclusions are that cutaneous and motor sensations play a very important rôle in the make-up of dreams, and that often in dreams that are predominantly visual the cutaneo-motor element is considerable, and may even incite visual hallucinations in accordance with the principle of sensory equivalence.

2. *Degeneration.*—This article is in the main historical. It deals with the works of Morel and Magnan on degeneracy, and the theories of Joffroy in the relation of muscular troubles to mental disturbances.

3. *Dementia Præcox.*—This article is a response of M. Klippel to the conclusions of M. Deny on dementia præcox, presented at the Congress of Pau. It is essentially controversial in nature.

4. *Sugar Superalimentation.*—This article is a plea for the freer use of sugar as an article of diet, both because of its nutritive value and because it is a powerful agent of calorification. The author recommends its addition in the proportion of 125 grammes to each litre of milk used in forced feeding. Sugar he thinks particularly valuable in the neuropathies, in all cases of emaciation, in tuberculosis, and in all who take violent or prolonged muscular exercise. It is preferably given in milk.

(November, 1904.)

1. Hospitals for the Reception of the Insane. MARIE.
2. Pseudo-Bulbar Paralysis. CHARPENTIER.
3. Conjugal General Paralysis. OLLIVIER.

1. *Reception Hospitals for Insane.*—This article is mainly historical, and constitutes a plea for psychopathic wards in general hospitals.

2. *Pseudo-Bulbar Paralysis.*—An account of a case presenting bulbar lesions, but on autopsy showing multiple areas of cerebral softening.

3. *General Paralysis.*—The record of a syphilitic who married, communicated syphilis to his wife, and both died of general paralysis.

(December, 1904.)

1. Experimental Psychology and Pedagogy. PIÉRON.
2. Note on a Case of Agitation Occurring as an Epileptic Equivalent. DAMAYE.
3. Application of the Method of Direct Observation in Experimental Psychology. LAHY.

1. *Psychology and Pedagogy.*—A critical review of methods of psychology, with suggestions as to their application in the education of abnormal children.

2. *Agitation and Epilepsy.*—This article is the account of a case of epilepsy occurring in a mental defective with convulsive and vertiginous seizures. These seizures disappear under treatment with bromide and hypochlorization, but a state of excitation, agitation, takes their place which itself disappears on the recurrence of the ordinary seizures.

3. This article is wholly psychological and does not contain much of interest to the psychiater. WHITE.

NEUROLOGISCHES CENTRALBLATT

(Vol. 23, June 1, 1904. No. 11.)

1. Concerning the Relation Between the Localization of the Convulsions and the Localization of the Anatomical Alterations in Experimental Tetanus. E. SJOVALL.
2. Concerning the Myelinated Fibers in the Cortex of Epileptics, Especially in the Outer Association Zones. T. KAES.
3. Concerning Fatigue Phenomena in Vibration. Preliminary Communication. W. NEUTRA.

1. *Experimental Tetanus.*—The author has previously reported alterations in the nerve cells in tetanus occurring in man. These alterations are by no means consistent, as other writers have reported different findings. Sixteen rabbits were tetanized. In nine the examination was completely negative, while in five the alterations were of different character. The nuclear changes consisted in a more or less diffuse change in the tigroid substance with alteration in the interstitial substance, and often a turgescence of the cell body. It was noted that the cell changes were found in the parts corresponding with the origin of the convulsion, but this was by no means always the case, for no cell changes were found in nine rabbits. The relation is not clear, but the whole process depends upon a physiological basis. If the nerve cells are sufficiently exhausted, changes occur; if not, none are found.

2. *Epilepsy.*—Kaes calls attention to a previous work of his in which he described a layer of association fibers which correspond to Meynert's second and third layer, in contradistinction to Bechterew's observation, who only described one such layer. Kaes found this in three epileptic brains, the patients having died in convulsions. He deduces no conclusions. He also observed that the cortex in epileptics is smaller than in the brain of others. This is different from the findings recently described.

3. *Fatigue.*—Neutra says that Erhard in 1872 used the vibration method as a diagnostic feature. The author, to determine the difference in vibratory sensation, first placed the fork, for instance, on the tibia, and when the vibration could not longer be felt, upon a symmetrical point on the other tibia. At the expiration of a few seconds this could not be felt. The difference in the vibration he called the "fatigue number." In hysterical or neurasthenic patients, should one fork be replaced on the first tibia it will be felt; or if, instead of the fork being placed on the second tibia, it is replaced on the first, it will also be felt. This is not present in normal individuals, and the author considers it a pathological factor, depending upon the fatigue of the individual.

(Vol. 23, June 16, 1904. No. 12.)

1. The True Centres of Motion. A. ADAMKIEWICZ.
2. Results of Examination of Liquor Cerebrospinalis. L. MERZBACHER.
3. A Contribution to the Posterior Regeneration in the Spinal Cord. G. BIKELES.

1. *Centers of Motion.*—If the cortex is removed from a lower animal it will be stupid, but will not have loss of power, but will have loss of

initiative. The author believes that the cerebrum controls only psychic functions, and that the true centers for motion are in the cerebellum. These have a definite center for certain motor functions, and are on the side of the inverted musculature. The musculature of the extremities has three centers. Each anterior and posterior extremity has its own, both anterior and both posterior identical centers, and all of the limbs a common center. The four extremities, therefore, are represented by seven centers.

2. *Liquor Cerebrospinalis.*—The author examined a large number of cases and in the main confirmed the findings of the French authors. A chemical examination did not show anything special. Lymphocytosis was found in tabes and general paresis, but not in diseases not syphilitic. It was also found in six epileptics out of twelve. This is a new finding. All forms of leucocytes were found.

3. *Regeneration in the Spinal Cord.*—Bikeles criticises the findings of Spiller and Frazier, who did not find a regeneration in the intramedullary portion of the posterior roots in an animal which had been operated upon ten months previously. Bikeles himself has found a regeneration, and asks for further information regarding the case of Spiller and Frazier.

(Vol. 23, July 1, 1904. No. 13.)

1. Ramón y Cajal's New Fibril Method. M. v. Lenhossek.
2. A New Flexor Toe Reflex. W. v. Bechterew.
3. Remarks upon Above Contribution. K. Mendel.

1. *New Fibril Method.*—This article is not suited for abstracting.

2 and 3. *New Reflex.*—Bechterew claims that he had demonstrated a new reflex which he called a "tarsophalangeal reflex," this being obtained by striking the dorsal part of the tarsal bone, there being a slight bending of the large toe. K. Mendel subsequently described the same reflex, not knowing anything of the first description.

(Vol. 23, July 16, 1904. No. 14.)

1. Concerning Compensatory Processes in the Human Spinal Cord. A. Pick.
2. Concerning the Cortical Innervation of the Bladder. L. v. Fränkl-Hochwart and A. Fröhlich.
3. Concerning the Association Fibers of the Small Cell Layer of the Cerebellar Cortex. L. Pussep.

1. *Compensatory Processes in the Spinal Cord.*—Pick records a case of infantile cerebral paralysis, in which the whole of one hemisphere is destroyed. The age is not known. It can readily be seen from the article's illustrations that the left half of the spinal cord is much smaller than the right. The posterior horns, however, are much hypertrophied on the affected side. Pick explains this by a compensatory hypertrophy due to the hypoplasia of the adjoining pyramidal tract. The injury must have occurred before the growth of the posterior horns was obtunded.

2. *Cerebral Innervation of the Bladder.*—The authors, after experimentation with lower animals, came to the conclusion that the primary act in the urinary function consisted in a relaxation of the sphincters and not in an overpowering of these muscles. In cutting the n. hypogastric and pudendi, a cortical relaxation may be apparent in the sphincters. These results are not constant, probably because the peripheral bladder innervation is not constant.

3. *Association Fibres in the Cerebellum.*—According to Bechterew, besides the ordinary processes, the small cells of the cerebellum have a collateral process which proceeds vertically to the upper layers. Pussep, by destroying the flocculus, was able to trace these fibers by the Marchi method Weisenburg (Philadelphia).

MONATSSCHRIFT FUER PSYCHIATRIE UND NEUROLOGIE

(Vol. 16, 1904, August.)

1. Experimental Studies Upon the Pathogenesis of Mental Disease. H. BERGER.
2. Recent Experiences Upon Mental Disturbances After Sulphide of Carbon Poisoning. F. QUENSEL.
3. Contribution to the Study of the Oculomotor Nuclei. G. PANEGROSSI.
4. Staining Methods. B. POLLACK.
5. The Twenty-ninth Travelling Assembly of the Southwest German Neurologists in Baden-Baden on the 28th and 29th of May, 1904. L. MANN.

1. *Mental Pathogeny.*—Berger describes his experiments in the production of neurotoxin. For this purpose he injected a goat with a fresh emulsion of brain substance obtained from dogs that had been bled to death. In this manner he was able to obtain a neurotoxic serum which was injected at first into the subdural sac of various dogs. Three died of meningo-encephalitis, which, according to Berger, was more severe than that produced by ordinary infection, and therefore represented the activity of the neurotoxic serum. Injected simultaneously, the results were various. In five dogs there were degenerative changes in the motor portion of the cortex; in two dogs no typical microscopical changes were observed. Three goats were injected, but on account of infection the results were negative. Dogs injected with normal goat serum showed no changes in the brain. A repetition of the experiment gave essentially the same results, and a myelotoxic serum was also prepared by injecting goats with an emulsion of the spinal cord. A series of experiments was then performed by injecting dogs subdurally with serum obtained from cases of dementia paralytica. A local infection was produced, but there were no evidences of progressive chronic inflammation. Other dogs were injected with the serum from various cases of dementia præcox, and showed some degeneration of the motor cells. A control dog inoculated with normal serum showed no change. In a series of 24 cases injected intraperitoneally changes were produced only in 5 cases. Some interesting experiments designed to elucidate various other phases of the subject were performed, particularly the mixture of sero-toxic substance and fresh brain substance before injecting, which resulted in marked weakening of the toxicity of the serum. Also an attempt was made to produce an anticytotoxic serum.

2. *Sulphide of Carbon Poisoning.*—Quensel continues the discussion of his two cases and calls attention to the neuropathic heredity in both, and discusses the possibility of some other factor than the carbon bisulphide, and also the differential diagnosis. He reports a fourth case which terminated fatally. The histological examination of the brain showed a diffuse degeneration of the ganglion cells in various parts. As the clinical symptoms had been those of acute delirium, and none of the ordinary pathologic changes of this disease were found, Quensel believes that an infection can be excluded. He gives the following conclusions: First, that carbon bisulphide may produce injuries to the brain. These may be either transient psychoses or even permanent defects. Second, that the diagnosis is to be made from the exposure to the poison, acute symptoms of intoxication before the outbreak of the psychosis, and an appearance of a series of somatic and nervous symptoms similar to those found in neuroses produced by carbon bisulphide poisoning.

3. *Oculomotor Nuclei.*—Panegrossi reports a series of studies upon the oculomotor nuclei of various species of monkey. The paper is still unfinished.

5. *German Neurological Society.*—The following papers were read: Remarks upon the Anatomy of the Human Cornu Ammonis, by Wiedersheim; The Occurrence of Natural Muscular Movements, by P. Grützner; The Alcoholic Febrile Delirium of Magnan, by Alzheimer; Comparative Psychiatry, by Kräpelin; Contribution to the Symptomatology and Anatomy of Spinal Apoplexy, by Dinkler; Periodic Paranoia, by Gierlich; The Coronal Fibers of the Lower Parietal Lobe, and the Sagittal Tracts of the Occipital Lobe, by v. Monakow; The Influence of Hunger and Sleeplessness upon the Cerebral Cortex, by Weygandt; The Behavior of the Abdominal Reflex in Disease of the Abdominal Organs, by F. Jamin; Investigations upon the Galvanic Light Reflex, by Bumke; An Epileptic Form of Cerebral Encephalitis, by Spielmayer; Diseases of the Anterior Cornua After Injury, by Hugo Stark. J. Sailer (Philadelphia).

(Vol. 16, 1904, September.)

1 Stereopsychoses. W. Alter.
2. Spastic Symptoms in Functional Disturbances of the Mind. A. Knapp.
3. Further Contribution to the Study of Oculomotor Nuclei. G. Panegrossi.

1. *Stereopsychoses.*—By stereopsyche Alter means the perception of form, distance, and relation in position: in other words, the perception of perspective through the correlation of the retinal image, accommodation and the movements of the eyeball. By stereopsychoses he means those mental disturbances which affect this perception of perspective. He has devised an ingenious diagram to represent the various factors entering into the stereopsyche. He discusses the various defects which are associated with stereopsychosis, and in analogy to the usual treatment of aphasia, classifies them according to the various hypothetical tracts that may be disturbed. He describes a case in a woman of 25, characterized by rapid disturbance of nutrition, and the development of a series of manifestations which could be regarded as functional disturbances resembling dyspraxia and dysgnosia. There is a long discussion of the mechanism by which these disturbances were brought about. which is not suitable for an abstract. He attempts to explain the stereopsychoses by analogy with the ion doctrine of chemistry.

2. *Spastic Disturbances in Functional States.*—A man of fifty-one years had for ten or twelve years occasional epileptic attacks. After one of these he became violently insane, and during the first days had increase in the reflexes of the lower extremities with persistent ankle clonus and the Babinski sign. These disappeared in a few days and the patient made a complete recovery, with amnesia for the period of attack. A woman of 46 developed pulmonary tuberculosis, in the course of which she had hallucinations. Ten days before death there were spasms in the arms and hypotonia in the legs. Two days before death there was some rigidity in the legs, associated with hypotonia. The reflexes were increased, but not pathological in type. Knapp gives a careful review of the literature of the reflexes in psychical conditions. In his second case he was able to determine the absence of any organic change in the brain.

3. *Oculomotor Nuclei.*—Panegrossi concludes his study of the oculomotor nucleus in the lower animals. The paper is not adapted to an abstract. J. Sailer (Philadelphia).

(Vol. 16, 1904, October.)

1. The Symptomatology of Atrophy of the Left Temporal Lobe. A. Pick.
2. Therapeutic Results in Acute Psychoses. F. Klein.
3. A Case of Combined System Disease Complicated with Bulbar Paralysis. R. Henneberg.
4. Two Rare Cases of Peripheral Nerve Palsy. Seiffer.

5. The Psychoses Occurring in Prisons. N. SKLIAR.

1. *Atrophy Left Temporal Lobe.*—A woman, 58 years of age, in addition to a chronic mental exaltation, developed an exquisite amnesic aphasia. Gradually she became word-deaf and died. At the autopsy there were no focal lesions, but an atrophy of the left temporosphenoidal lobe. The pathological findings seemed to confirm the idea of Quensel that in these cases the lesion is situated in the second and third convolutions. An additional lesion in Broca's convolution sufficed to explain the disturbance of speech. Another case, a woman of 75, whose language was entirely confused, did not appear to understand what was said to her. She understood mimic gestures and recognized objects. There was general atrophy of the brain, especially in the neighborhood of the left temporal lobe. The case represents amnesia, agrammatism, paraphasia, agraphia, and alexia without object blindness, associated with progressive dementia. The case merely indicates how manifold are the symptoms following atrophy of the temporosphenoidal lobes. The third patient, a woman of 38, with distinct signs of constitutional syphilis and commencing paretic dementia, and with some paralysis of the ocular muscles, had amnesic aphasia. There was general atrophy of the brain, particularly marked in the left temporal lobe, and some thrombosis of the veins. It appears reasonable to consider that the aphasic symptoms were associated with more pronounced atrophy of the temporal lobe.

2. *Therapy in Acute Psychoses.*—Klein discusses certain drugs used frequently in the treatment of mental conditions. Hyoscine hydrobromate he regards as extremely dangerous. He gives the clinical reports of thirteen cases of various forms of insanity treated by this agent, with the production of hyoscine delirium. The symptoms are paresthesia, visual hallucinations, and occasionally disturbance of the other special senses. The deliriums usually occur when awakening from the narcosis. It is, therefore, a dangerous remedy, but is to be used in all cases in which it is necessary to quiet the patient rapidly. Trional is a valuable remedy, acting particularly well in depressive states. It should not be given continuously in small doses. Sulphonal, properly speaking, is a sedative, less valuable as a hypnotic because not as trustworthy. It is most valuable in the chronic psychoses, but occasionally produces disagreeable effects. Morphine is less used than formerly. It is chiefly valuable in psychoses of anxiety, and in these conditions is far more effective than any other remedy. In the most severe forms of melancholia it seems to have an almost curative effect, and there seems to be some advantage in combining it with trional. It should be employed subcutaneously.

3. *Combined System Disease and Bulbar Paralysis.*—A woman 38 years of age, after some gastric disturbance, developed weakness and paresthesia in the legs, bulbar symptoms, and disturbance of micturition. The paresis of the lower legs continued to progress, but the bulbar symptoms improved. Later there was atrophy of the leg muscles, and some atrophy of the hand muscles, with disappearance of the patellar reflex. The disease progressed slowly. In seven years there was moderate dementia, bulbar dysarthria, paresis and atrophy of the tongue, weakness of some of the muscles of the mouth and jaws, of the vocal organs and of some of the muscles of the eyeballs. The pupillary reactions persisted. Still later there was disturbance of the muscular sense in the legs, diminution in the electrical irritability, with partial reaction of degeneration, lancinating pains and girdle sensation, with paresis of the diaphragm. There appeared to be disease of the pyramidal tracts and posterior columns of the cord, and of the anterior cornua of the gray matter, and in addition the symptoms of bulbar paralysis. At the autopsy there was found degeneration of the pyramidal tracts, the cerebellar tracts, the posterior columns, disappearance

of the ganglion cells in Clarke's columns, in the anterior cornua and in the nuclei of the medulla. Henneberg collects from the literature some cases showing more or less similarity to his own.

4. *Peripheral Nerve Palsy.*—Seiffer reports the case of a young girl who in childhood had had symptoms of irritation and paresis in the region of the right brachial plexus. These symptoms gradually progressed until, when she was 17 years of age, they impaired her general health and interfered with the use of the right arm. The Röntgen ray showed the presence of a cervical rib on the right side. This was removed by operation, and the subjective symptoms of the patient were greatly improved, but the motor symptoms remained about the same. Previous to the operation, if the arm was raised or the head turned the radial pulse disappeared; after the operation these symptoms no longer existed. The second case, a musician 52 years of age, had an isolated paralysis of the musculo-cutaneous nerve, evidently due to neuritis, and probably in part superinduced by the severe work to which this particular nerve was subjected in the course of his profession. There were two symptoms of interest: first, the proof that the brachialis internus was supplied by two nerves, a branch from the musculo-cutaneous and one from the radial; second, when Erb's point was stimulated only the deltoid responded. Only from twelve to fourteen cases of isolated paralysis of the musculo-cutaneous nerve have been reported, and only two other cases in which the paralysis was due to neuritis.

5. *Psychoses in Prisons.*—Skliar, after discussing the statistics of insanity among prisoners and the various forms that may occur, reports his own cases. These were: 5 of delirium tremens; 21 of acute psychosis; 21 of dementia; and 15 of paranoia. The paper is still unfinished.

J. Sailer (Philadelphia).

MISCELLANY

Further Notes on the Treatment of Birth Paralysis of the Upper Extremity by Suture of the Fifth and Sixth Cervical Nerves. Robert Kennedy (British Medical Journal, Oct. 22, 1904).

In this paper Dr. Kennedy completes the histories of the cases reported by him in the *British Medical Journal* in 1903, and reports two additional cases. In the first of these a typical Duchenne's paralysis of the right arm was noticed the day after birth. No improvement having occurred two months after birth, an operation was performed in which the junction of the fifth and sixth cervical nerves was found to be cicatricial. Excision with suture was done as in the previous cases. Improvement began in about three months, and in fifteen months restoration of the functions was perfect except in the supinators. The second case was very much like the first, except that the extensors of the hand were also involved. An operation similar to the foregoing was followed by improvement beginning four months after operation. A year later improvement was marked. The lesion in these cases is produced by a stretching of the nerve cords during delivery, both the fifth and sixth cervical nerves being involved. C. D. Camp (Philadelphia).

A Case of Hemorrhage Into the Lateral Ventricle. E. L. Pope (British Medical Journal, Dec. 31, 1904).

A woman, aged 24 years, one month after a slight concussion of the brain developed an intense headache and vomiting. She then became stuperous and for the next twenty-four hours had gushing vomiting at intervals. There were no localizing signs and no paralysis. The optic discs were swollen and pale and there were numerous retinal hemorrhages. Later she had rigidity in one or two limbs at a time, followed by a relaxation in these limbs and rigidity in the others. The temperature was subfebrile, respirations 26, pulse 120. At autopsy there was found a recent hemorrhage into the right lateral ventricle and into the third ventricle through the foramen of Munro. In addition there were two small hemorrhages, about the size of a marble, into the brain substance, one in the central part of the left frontal lobe and the other in the central part of the right occipital lobe. There were no signs of fracture of the skull or of a meningitis. C. D. CAMP (Philadelphia).

INTERMITTENT EXOPHTHALMOS W. C. Posey (Journal A. M. A., Feb. 18).

Posey reports an instance of this rare affection, of which he is able to collect only 39 cases in the literature. He thinks that it is probably more frequent than supposed. Its characteristic symptom is the pushing or falling forward of the eyeball when in a dependent position or when the return of the blood from the head is interfered with by holding the breath, pressure on the jugulars, coughing, sneezing, tight collars, etc. The proptosis is usually unaccompanied by pain and the patient may be unconscious of it. Vision may be unaffected or permanently impaired or lost. Generally it is impaired during the protrusion. The diagnosis is easy and the prognosis is generally good. The patient should avoid excessive strain and anything that causes the eye to proptose. Ordinarily operation should be advised against, though where hemorrhage has occurred and vision is threatened Hitschmann's counsel to lay bare and resect the affected veins may, perhaps, be followed, Krönlein's operation being resorted to if necessary.

LOCOMOTOR ATAXIA. Guy Hindsdale (Journal A. M. A., Feb. 18, 1905).

The author reports an apparently typical case of tabes without positive syphilitic history treated by rest, massage, electricity and educational movements, and internally, nitrate of silver and extract of belladonna (as there was intolerance for the iodides). The symptoms generally disappeared. In eight weeks there was an increase of nearly forty pounds in weight. The urine, which had contained albumin and casts, became normal, and in three months he returned to his former occupation apparently well. After three years of usefulness he came again under treatment with similar symptoms. His weight was reduced to 104 pounds. The same treatment, with the addition of hypodermic administration of mercuric chlorid, 1-20 grain twice daily, was employed, with like results as in the first attack. In nine weeks he had gained forty-six pounds. The case is reported as of interest in showing the value of the rest treatment when employed sufficiently early with electricity and massage or exercise. While a cure is not claimed in the case, there is at latest reports improvement in all lines.

SPASTIC DIPLEGIA DURING PERTUSSIS. J. H. W. Rhein (Journal A. M. A., March 4, 1905).

The case of a child, thirty months old, is here reported, who had spastic diplegia in the legs with nystagmus. Later a similar condition of the arms gradually developed, with difficulty in swallowing, etc.The child became greatly emaciated and finally died after numerous general convulsive attacks, worse on the right side. The autopsy revealed numerous small hemorrhages in the right and left frontal cortex, slight thickening of the pia in the paracentral region, with a few small cortical hemorrhages. There were areas of distended perivascular spaces containing many round mononuclear cells, which were also distributed everywhere throughout the cortex; their protoplasm was granular. In the occipital region there was thickening of the pia and intense red blood cell infiltration. Microscopically, marked degeneration was observed in one cerebral peduncle.

The general appearance was that of a hemorrhagic meningo encephalitis. Rhein believes that the widespread lesion of the cortex was due to a toxin acting on the vessels and setting up an inflammatory process. This caused local destruction of fibers, especially in the paracentral region, followed by degeneration in the pyramidal tracts. The minute cortical hemorrhages he attributes to the convulsive attacks preceding death.

The Treatment of Cerebral Tumors. Brunniche (Graduation Thesis, Copenhagen).

This work consists of a careful review of some 208 cases of cerebral tumors. Of these 17 were tuberculous, 11 syphilitic, and 125 various forms of new growths. The diagnoses were verified either by operation or necropsy. In only 14 cases could the tumor be localized sufficiently to permit of operation being advised. In 2 of these radical operations were undertaken, and 1 patient was cured, while the other was considerably benefited. In 4 cases palliative operations were undertaken, and of these 2 were much improved, 1 living for 7 years after the operation. Brunniche considers that with improved technique and better localization of the tumors, operation offers a degree of success which medical treatment cannot attain.

Jelliffe.

Multiple Neuritis. Wharton Sinkler (Journal A. M. A., Feb. 25, 1905).

The author, after discussing the various causes of multiple neuritis, such as alcohol—by far the most frequent—coal-gas poisoning, carbon disulphid, metallic poisons, white lead, copper, phosphorus, mercury, etc., calls attention to the use of patent medicines containing alcohol as a possibility. He reports a case due to the use of arsenic as a medicine in a child treated for chorea, and refers also to the epidemic traced to arsenic in the glucose used for making beer, which was reported in England in 1899. He also refers to infectious diseases as a cause of this condition and reports four cases from an apparently hitherto unrecorded cause, namely, puerperal septicemia. In conclusion, he reports a case of unknown origin, one of a class that is rather difficult to diagnose from Landry's disease except by the later involvement of the bulb in the latter affection.

Hemiplegia. T. H. Weisenburg (Journal A. M. A., Feb. 25, 1905).

Dr. Weisenburg has studied 160 cases of hemiplegia in the Philadelphia General Hospital with special reference to heredity, pain, muscular atrophies, respiration, edema and the arthropathies, vasomotor disturbances and hemichorea. In 109 cases where the facts could be ascertained heredity was present in 14 and strongly manifested in 5 cases. In 17 there was a prehemiplegic pain, which is accounted for as possibly due, in persistent cases, to cerebral congestion or actual small hemorrhage in the sensory pathway. Twenty-seven had post-hemiplegic pains. In 30 cases there was either total or partial anesthesia, and in the majority some pain. It appears that pain in hemiplegia is most likely to occur in cases with sensory changes. Weisenburg confirms Hughling Jackson's observation of the great expansion of the upper portion of the chest on the paralyzed side during quiet respiration, but he also found, and Dr. Spiller confirmed the observation, that at the end of ordinary or quiet respiration the chest retracted more than on the paralyzed side, thus showing greater power of expelling the air on the sound side and actual weakening of the lung of the affected side with diminished respiratory movement. He reports a case of intense edema on the paralyzed side; in two other cases there was edema of the paralyzed hand. Among vasomotor disturbances he notices the rare occurrence of anhidrosis in one of his patients. Weisenburg considers

Bonhoeffer's explanation of a lesion in the extension of the anterior cerebellar peduncle into the subcortical ganglia as the most probable theory concerning posthemiplegic chorea. In every one of his 160 cases there was some muscular atrophy, more marked as the paralysis was marked, affecting the upper more than the lower limb and, in the majority of cases, accompanied with sensory symptoms. In the large number considerable atrophy was also observed on the so-called sound side. Arthropathies, as described by Marie, occur in most cases of hemiplegia. They affect mostly the shoulder joint, but with marked contractures other joints also may be involved. Weisenburg thinks the cause is probably the forced immobility plus the pulling on the articulations and tendons by the weight of the paralyzed member. Lesions of cells of the anterior horns are not common in hemiplegia and, in the cases examined, no pathologic changes were found here. He notes, however, one or two peculiar cases, and in two or three of his patients there were painless arthritic conditions suggesting somewhat the arthropathies of chronic spinal disease. This paper is to be followed by a pathologic study of hemiplegia.

LOCOMOTOR ATAXIA. J. W. Rhein (Journal A. M. A., Feb. 25, 1905).

In this the author reports a case of locomotor ataxia with a typical clinical history, save that there appeared with or soon following the appearance of the tabetic symptoms a fine rhythmical tremor in both hands and arms. This was quieted by voluntary motion, but afterward increased. No other symptom suggesting paralysis agitans was present, and the pathologic findings at the autopsy were those characteristic of well-developed tabes. The case is of interest on account of this occurrence of tremor, which is not usually observed as a symptom of tabes. The author suggests that it may possibly be a case of this disease associated with paralysis agitans. The arterial conditions were not such as are usually associated with senile tremor.

A CASE OF LANDRY'S PARALYSIS. C. H. Foley (British Medical Journal, Jan. 7, 1905).

An agriculturist, aged 35 years, of temperate habits, first noticed that his feet felt benumbed and weak. A few days later his lower limbs were completely paralyzed, the paralysis rapidly extending to his hands, arms and trunk. Sensation was dulled, but there was no anesthesia. The paralysis was flaccid and the tendon and superficial reflexes were lost. Deglutition was noisy and spasmodic, and constipation was absolute for some days. There was tenderness over the mid-dorsal spine and in the muscles of the limbs on deep pressure. Treatment consisted of the administration of a saline diaphoretic, containing 20 minims of ferric chloride to each dose, every four hours. The spine was blistered along its whole length and later massage was given twice daily. He recovered gradually and was completely well in two months, although the knee-jerks remained diminished to some extent.

C. D. CAMP (Philadelphia).

THE PAINS OF TABES. Sir Wm. R. Gowers (British Medical Journal, Jan. 7, 1905).

In a clinical lecture on this subject, the author discusses in detail the symptom of pain occurring in tabes dorsalis. He divides the pains according to their character, into two classes. In Class A are those in which the pains are very brief and succeed each other, after intervals, in the same place. These pains are further divided into superficial and deep. The superficial pains are commonly referred to as "lightning pains," and leave the skin area involved sore to touch. The second variety of the brief pains is deep seated, but not referred to any definite structure. Class B includes those that are prolonged, sometimes hours or more, of varying intensity and with remissions, but not intermissions. They are more commonly truncal, but may affect any part of the body, and their diagnosis from local organic disease is very important. A subdivision of the second class consists of widely diffused sensations, such as cold, heat, swelling, etc., seldom amounting to real pain, but distressing on account of their persistence Under the heading, "Tabetic Neuralgia," the author summarizes the symptoms of eleven cases which form in his experience a special variety of tabes, in that the pains are the only prominent symptom and the knee jerks are not lost. He assumes that there is a difference in the casual agent, the toxin, in these cases, as there is when the toxin has a special tendency to act on the optic nerves. All these cases were in adult men with a history of syphilis in 9 of the 11, and an average interval between the primary disease and the onset of pain of 12 years. The knee jerks were normal in degree and equal on the two sides in 8 out of the 11 cases. In two cases they were unequal, but in only one was there a lost reflex, and then only on one side, the other side remaining normal. In two cases the light reaction of the pupils was lost, in two others it was slight, but present, and in two it was completely lost only on one side. One case presented characteristic optic nerve atrophy. Sensation was quite normal in most cases. The pains presented many of the variations met with in ordinary tabes, and were generally severe. The inference that the extremities of the peripheral nerves is the source of the tabetic pain agrees best with the known facts concerning them, though other portions of the nervous system may play a part in their production. For the relief of the pain the coal tar derivatives are of most value when the pains are of moderate intensity. They are most effectual when given in one large dose at the onset of the attack. In later and more severe cases morphine must be used. The tendency to the attacks may be relieved by the chloride of aluminium, in doses of 5 to 10 grains, three times a day, and a similar influence may sometimes be obtained with the salicylate series of drugs.

C. D. Camp (Philadelphia).

Muscle Atrophy with Tabes J. Hagelstam (Finska Läkaresallskh, 45, p. 635).

The author reports the case of a man without bad heredity; possible lues and alcoholic history. At the age of 16-18, having lifted heavy weights, strained himself, and was also much exposed to cold weather. From the time when he was 18 years old he noted a marked grade of weakness of the arms. Later a similar weakness of the legs and thighs developed. At the age of 20 many of his teeth dropped out, and at the same time he had attacks of diplopia. These were helped by the use of glasses. In late years, inability to hold the bladder contents, weakness of the anal sphincter and shooting pains in the legs. Examination showed marked muscular atrophy of the muscles of the shoulder, the right upper arm and thigh. The forearm and hand showed no atrophy. The legs were thinned, in extreme hypertension, marked hypotonus, Patellar reflex lost, Achilles and abdominal reflexes active; no ataxia in reclining position. No Romberg, papilla pale gray; acuteness of vision and visual fields not modified; diminished sensibility of the toes and also some change in position perception. The case is interpreted as one of mild involvement of the combined posterior columns with progressive muscular atrophy.

Jelliffe.

A NOTE ON SENILE SYMMETRICAL ATROPHY OF THE SKULL. F. Parkes Weber (British Medical Journal, Jan. 21, 1905).

The process is probably due to lacunar absorbtion accompanying senility or other chronic states of depressed nutrition. Not every senile change is present in every senile person, and some develop relatively early on account of special predisposing factors, such as hereditary, wasting diseases, or chronic causes of depressed nutrition. The lacunar absorbtion which causes the atrophy of the outer table in the parietal bones is probably not due solely to senility, but is like other senile atrophies, intimately connected with it. The localization of senile atrophies is probably to be accounted for by portions being selected which are no longer much used, and which are of least vital importance, thus the parietal bones atrophy at a site which is not pulled upon by muscles and is of comparatively little importance for maintaining the strength of the brain case and its safety from concussion.

C. D. CAMP (Philadelphia).

ALCOHOLIC BORDERLINE PSYCHOSES. F. P. Norbury (Journal A. M. A., March 18, 1905).

Norbury gives the history of two cases of alcoholic mental derangement: one of typical alcoholic delusions of infidelity following the stoppage of alcohol in a steady drinker, and the other of acute alcoholic maniacal attacks following excesses. Both cases are discussed at some length. His conclusions seem to apply more particularly to the latter type, which he considers as properly classed among the minor psychoses of alcoholism. He says that from an experience with several hundred such cases, including the polyneuritic psychoses marked by amnesia, paramnesia and confusion, with marked hallucinations, he has been led to the following conclusions regarding these mental disorders: 1. They are rare in acute alcoholism, but may appear in adolescents of neurotic type. 2. They are more frequent after adolescence and up to 40 or 45 years of age. 3. They both occur in continuous drinkers and in periodic delinquents. 4. The prognosis is variable, depending on inherited frailties and on moral development. 5. Early treatment is advisable as a prevention of major psychoses. 6. Treatment is successful in the majority of cases, providing the physician has the earnest co-operation of the patient and can have him under his care for a protracted period. 7. Such cases should be distinguished from ordinary chronic alcoholism and should be treated from the standpoint of mental diseases.

LARGE TUMOR OF FRONTAL LOBE. Philip King Brown and W. W. Keen March 11, 1905).

The authors report a case of an immense tumor (angiosarcoma) of the frontal lobe. The symptoms were insidious, there was very little pain, but some mental impairment and later blindness. There was also disturbance of the olfactory sense, exophthalmos and other symptoms indicating localization. The operation involved a removal of bone for a circumference of 37 cm. in the left frontal region. The tumor extended back to the limits of the incision; it had eroded the posterior wall of the frontal sinus, the orbital plate and the two plates of the frontal bone, as far back as the posterior limit of the frontal. Notwithstanding the pressure on the nerves of the eye and its muscles and on the eye itself, a single dose of five grains of phenacetin controlled the only pain of any note from first to last.

MYXEDEMA AND DIABETES MELLITUS. A. A. Strasser (Journal A. M. A., March 11, 1905).

The author reports the case of a child, 8 years old, in whom the characteristic symptoms of myxedema appeared after weaning. The thyroid treatment was instituted with marked improvement in the symptoms, but diabetes intervened and it was discontinued, not because it was considered responsible for the intervening condition, but to eliminate it as a possible factor. The case was very carefully studied as to its metabolism; the child improved greatly in its mental symptoms as the diabetes progressed, but finally died in coma and convulsions. The author discusses the case with special reference to the effect of the diabetes on the myxedema, and considers the case as absolutely unique. Diabetes mellitus itself is not so rare in children as was formerly thought, but its occurrence in myxedema with the apparent marked effect on the latter condition here observed has not been reported heretofore. In a supplementary note he refers to two somewhat similar cases reported by Dr. Alfred Gordon in *American Medicine*, Feb. 6, 1904, but he does not agree with the optimistic Gordon views as to the prognosis in such cases.

THE CONDITION OF FEAR IN CARDIAC DISEASE. W. R. Denton, Jr. (Journal A. M. A., February 18).

Incited by a statement that all cases of alienation showing apprehension revealed cardiac diseases, the author examined the twenty-five most recent admissions in the Sheppard and Enoch Pratt Hospital, and some twenty cases in the Johns Hopkins Hospital. He concludes that the cardiac lesion is not the primary factor in causing the associated state of apprehension. What we may call, for want of a better term, the idosyncrasy of the patient, is largely responsible for apprehension associated with cardiac lesions. In neurasthenic types a lack of vagus control is an important etiologic factor. Our knowledge of the subject is still not yet exact, and he asks the co-operation of clinicians in the investigation.

DELIRIUM AS A SYMPTOM OF HYSTERIA. Theodore Diller (Journal A. M. A., March 4).

While it is well known, writes Dr. Diller, that delirium constitutes the final phases of classic attacks of major hysteria, the subject of insanity in relation to hysteria seems to be in a state of more or less confusion or doubt. This, he believes, is due to the fact that these cases are not studied in their whole course by the same men. The family physician sees the earlier symptoms and the asylum physician the later manifestations. Diller says that some authors call certain mental symptoms hysterical when they are mild or of short duration, but apply the designation "insanity" to the same symptoms when they are prolonged beyond a certain time or when they become more striking in type. He quotes at some length from Janet on the subject of delirium as a symptom of insanity, and reports several illustrative cases. In one case a diagnosis of typhoid was made, but Diller considered it a secondary infection, favored by the condition of the patient. In all the cases reported there was more or less marked neurotic disturbance or shock, and he considers that the diagnosis of hysteria was justified.

PARALYSES OF THE BRACHIAL PLEXUS. Henri Grenet (Gazette des Hôpitaux, Sept. 24, 1904, No. 106, and October, 1904, No. 112).

In these papers the author gives a full account of the paralyses of the brachial plexus. After an anatomical description of the formation of the plexus, a clinical division is made into paralyses from lesions of the roots and of the plexus proper.

Etiology. This is classed as traumatic and nontraumatic.

I. Traumatic.

1. Wounds of the supra-clavicular region, caused in various ways; sometimes the resulting paralysis is not caused directly by the

wounding agent, but by resulting inflammation, especially of bones.

2. Other forms of trauma usually affecting the roots; these are falls upon the shoulder, injury by weights falling on the shoulder, prolonged maintenance of certain positions of the arms, such as behind the head during sleep and in the administration of anesthetics, dislocation of the shoulder and manipulations in reduction, and notably in difficult labors (Duchenne's obstetrical palsy).

II. Non-traumatic.

1. After some infectious fever.
2. Apoplectiform neuritis of Remak and Dubois.
3. Diseases of the meninges and vertebral column.
4. Tumors in the supra-clavicular region.
5. Aneurysms of sub-clavian and carotid.
6. Supernumerary rib.

Pathogeny of the paralyses caused by indirect violence.

Many explanations have been given of the mechanism of production. In dislocation it is said that the plexus may be directly compressed by the head of the humerus. Against this view it is objected that the head of the bone is not always in contact with the nerves, and that from the position of the displaced humerus we should expect paralyses of the nerves of distribution and not of the plexus itself. A violent blow on the shoulder is thought to cause compression of the plexus between the clavicle and the first rib. Evesque suggests a double mechanism; first, the head of the humerus forcibly flattens the plexus against the ribs, causing a contusion; secondly, the clavicle is elevated and compresses and contuses the roots against the transverse processes. In violent blows on the scapula this bone is depressed and the plexus is thought to be compressed between the first rib and the clavicle; the same compression might occur when the arm is elevated. Others, however, maintain that in elevation of the arm the roots are compressed between the clavicle and the transverse processes of the last cervical and first dorsal vertebrae. The obstetrical palsy has been said to result from compression of the nerves at the point of Erb by the forceps. A more probable explanation of this variety of paralysis, and one also applicable to other forms, is that it is caused by forcible inclination of the head to the opposite side at the same time as there is depression of the shoulder. This view was put forward by Fieux from experimental evidence, and has been adopted and amplified by Duval and Guillain, who state that forcible depression or elevation of the shoulder may cause rupture of the roots of the plexus, whilst less severe injury may cause merely tension or partial rupture.

Pathological Anatomy. The roots have been found torn away from the cord; atrophy of the cells of the ventral coruna has been found; the 6th and 7th roots have been found embedded in inflammatory tissue at the level of the transverse processes; the 5th root has been found ruptured. Déjerine has found a hemorrhage in the sheath of the plexus proper in a case of apoplectiform neuritis.

Symptoms. 1. Total Radicular Paralysis.

(*a*) Motor. Total flaccid monoplegia, the arm hanging helplessly by the side and in the position of internal rotation, with some elevation of the shoulder, attributed either to instinctive contraction of the upper part of the trapezius or paralysis of its lower part (Guillemot). The electrical reactions are altered.

(*b*) Sensation. Anesthesia of all forms of sensation, or to pain and temperature alone, occupying usually the hand and forearm, or in addition the outer and posterior surface of the arm. The internal surface is sentient, since the cutaneous nerves of the plexus which supply it also receive branches of the 2nd and 3rd dorsal. There may, however, be disseminated islands of anesthesia in this region.

(*c*) Oculo-pupillary signs. Narrowing of the palpebral fissure and contraction of the pupil, if the lesion is situated proximal to the point of exit of the white ramus of the first dorsal nerve.

(*d*) Trophic and vasomotor disturbance.

2. Superior Radicular Paralysis (Duchenne-Erb). Limited to the 5th and 6th cervical nerves. A paralysis originally total may be finally limited to these nerves, or they may from the first have only been affected. The muscles constantly paralyzed are the deltoid, infraspinatus, biceps, brachialis anticus and supinator longus. Other muscles may be paralyzed. (See the recent works of Wilfred Harris and Kennedy.)

Sensory anesthesia is usually absent, if sought for some time after the onset, but is present at first. Vasomotor and trophic disturbances are occasionally found.

3. Inferior Radicular Paralysis. Results from a lesion of the 7th and 8th cervical and 1st dorsal nerves. The motor paralysis is in the muscles supplied by the outward median nerves, producing weakness and atrophy of the small muscles of the hand. Anesthesia is the rule occupying the internal part of the hand and forearm. Oculo-pupillary symptoms are characteristic, but may be absent. When present they show that the lesion is within the vertebral canal. Trophic and vasomotor disturbances are more common than in the superior radicular paralysis.

4. Complex Radicular Paralysis. A partial lesion giving a more irregular distribution of paralysis, motor or sensory, may occur. In such cases, if there is paralysis present, either of the serratus magnus, supra- or infra-spinatus muscles or oculo-pupillary symptoms, the lesion is radicular. If these signs are absent, it may be impossible to localize the site of the lesion.

5. Uniradicular paralyses affecting either the 5th cervical or 1st dorsal have been described by Charcot and Farquhar Buzzard.

6. Purely motor paralysis may occur, since the evidence shows that the anterior roots are more readily injured than the posterior.

7. Purely sensory paralyses are very rare. Raymond has recorded an example in which a hemipachymeningitis invaded only the posterior roots and brachial plexus. A lesion limited to one or two roots gives rise to either very slight anesthesia or none at all.

Paralysis of the Plexus Proper.

The resulting paralysis either resembles those of radicular origin or follows the distribution of the peripheral nerves. A paralysis is radicular if oculo-pupillary symptoms are present, or if the serratus magnus, supra- or infra-spinatus muscles are paralyzed, otherwise its exact site cannot be indicated.

Observations on the Forms of Paralysis due to varying etiology.

1. Traumatic. Here the paralysis is sudden. Spontaneous pain may be present at first, but tends to disappear. The paralysis may at first be total, but later limits itself to upper or lower type. The anterior roots are usually more injured than the posterior, and sensory paralysis tends to disappear.

2. Obstetrical. Oculo-pupillary symptoms may be present, but are usually absent. A more severe lesion follows breech than head presentations.

3. Intra-vertebral Compression. Usually from pachymeningitis, often tuberculous, tumors, etc. Violent pain often precedes the paralysis for a long time. Later symptoms of pressure on the cord appear.

4. From nereditary syphilis, either of the upper or lower type. They are cured by mercurial treatment, and are due to meningo-myelitis

5. From extra-vertebral compression. Mode of onset varies according to the nature of the compressing agent. When this is an aneurysm Duval and Guillian consider that the paralysis is not due to simple compression, but to the inflammatory formation around the walls of the aneurysm, and that hence the paralysis may be permanent even if the aneurysm is in process of healing.

6. Apoplectiform Neuritis. The onset is necessarily sudden.

Diagnosis. In infants, syphilitic pseudo-paralysis, which appears some months after birth, osteo-myelitis and infantile paralysis have to be considered.

In adults, the diagnosis of hysterical brachioplegia may be difficult, since it has been considered that this may occur even when unassociated with anesthesia. Cortical lesions are not likely to be mistaken for those of brachial plexus, and are distinguished by the presence of conartcture, exaggerations or reflexes, absence of wasting and of sensory disturbance.

Of spinal disease, acute anterior poliomyelitis, syringomyelia and hematomyelia must be considered; the latter is rarely limited enough to produce only a brachial monoplegia. Sometimes a violent tearing of the roots may give rise to hematomyelia above and below the radicular territory, and then analgesia and thermoanesthesia would be found.

Amyotrophy from joint disease can be excluded by the history and the absence of altered electrical irritability associated with increase in the tendon-jerks.

Prognosis. This varies greatly, according to the violence of trauma.

Destruction of the cells of the ventral horn may follow, and even a limited hemorrhage into the cord. The condition of the electrical reactions gives some indication of the probable duration of the paralysis.

Treatment. Massage, electricity and hydropathy are required. Surgical interference may be necessary. [The deformity produced by paralyzed muscles must be corrected by the use of a sling to support the forearm when the biceps group is paralyzed, or by a light metal splint applied to the anterior surface of the forearm, reaching as far as the end of proximal phalanges and bent backwards to support the hand when the extensors of the wrist and fingers are paralyzed. By thus removing tension from the weakened muscles recovery is largely aided.]

W. B. Warrington.

NEURO-FIBRILS AS DISPLAYED BY THE METHODS OF PROF. RAMON-Y-CAJAL.

L. Azoulay (La Presse Médicale, July, August, September and October, 1904).

Dr. Azoulay's review of Prof. Ramon-y-Cajal's observations shows, apparently, that the presence of a neuro-fibrillar network in the interior of nerve cells has at last been definitely established. The earliest stages of its growth have not been ascertained, but there seems to be little doubt that it commences to grow first in the dendrites, and progresses thence into the interior of the cell body, where it evidently differentiates into a series of primary and secondary fibrils. The former are most numerous in the perinuclear network, and the latter in the more peripheral parts of the cell, though they are found in all parts, intervening between and apparently connecting the primary fibrils together. The network formed by the two sets of fibrils may be distinctly recticular or more or less fascicular, the former being the more primitive and the latter the more advanced condition; but distinctly as it appears, the network is not an unchangeable entity, for it undergoes remarkable alterations associated with the activity and quiescence of the cells. Thus in hibernating animals the fibrils of the intracellular reticulum become fewer and larger, the finer fibrils disappearing, and on the larger remaining fibrils there are numerous spindle-shaped enlargements. Similar appearances are presented by the nerve cells of rabbits in the paralytic stage of hydrophobia. If, however, a hibernating animal is exposed to a temperature which revives it and induces it to pass from a state of lethargy to a state of activity, then the finer fibrils reappear in the intracellular reticulum. It follows, therefore, that the appearance of the reticulum, as demonstrated by the reagents employed, is merely the expression of the condition of activity of the cells at the time of their death and fixation.

JELLIFFE.

SURGICAL TREATMENT OF EPILEPSY. Delegénière (Arch. Prov. de Chir., No. 10, 1904).

This author advocates a novel treatment of epilepsy by ligature of the superior longitudinal sinus just above the torcular Herophili. This method of dealing with epilepsy was suggested to the author by the very good results observed in a case of this disease in which, during an operation for trephining, it was found necessary on account of an accidental wound to apply a ligature around the longitudinal sinus. Regarding an epileptic attack as a clinical syndrome common to several pathological conditions, and holding that the various causative lesions of epilepsy tend in different ways to produce the common result of permanent dilatation of the veins of the external surface of the cerebral hemispheres, Delagénière considers it a logical step to attempt to modify the venous circulation of the cerebral cortex by practicing ligature of the terminal portion of the longitudinal sinus. Under the assumption that distention of the superficial cerebral veins is probably the cause of irritation of the cortical layer of the brain, he would endeavor to overcome this venous distension by an operation analogous in its aim and mode of action to ligature of the upper part of the internal saphena vein for varicosity of the veins of the leg. The object in practicing ligature of the longitudinal sinus would thus be to bring about a collapse of the over-distended veins by which this sinus is supplied with blood. The author describes the different stages of this operation, which he has performed in a single case with good results. The longitudinal sinus is exposed by trephining the cranial vault in the posterior half of a line drawn from the bregma and the external occipital protuberance, at a point about 8 cm. or 9 cm. behind the bregma.

JELLIFFE.

Book Reviews

MORPHIUM ALS HEILSMITTEL. By Professor D. O. Rosenbach of Berlin. Fischer's Medicin. Buchhandlung, H. Kornfeld, Berlin.

In this brochure, about 100 pages, Dr. Rosenbach points out the general principles of therapy which should guide one in the use of various narcotic remedies, particularly morphine.

He discusses euthanasia, the value of morphine in organic heart diseases. its tonic properties, its use in nervous disorders, in febrile conditions, in pneumonia and congestive pulmonary states, cough, phthisis, hemoptysis, uremia, and finally the dangers of the development of the habit.

There is some doubtful pharmacology in the author's statements. He apparently has not followed this branch of work. There is also a general air of positivism and a dogmatic form of expression which makes an unpleasant impression on the reviewer, but there are many points of interest and of value in the work. There is little very new, and what is new, if we may be permitted to quote, is not always true, yet all in all, the dissertation may be said to be worth while. JELLIFFE.

THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS. Edited by Gustavus P. Head, M.D. Volume VIII, Materia Medica and Therapeutics, Preventive Medicine, Climatology, Suggestive Therapeutics, Forensic Medicine. Edited by George F. Butler, Ph.G., M.D.; Henry B. Favill, A.B., M.D.; Norman Bridge, A.M., M.D.; Daniel R. Brower, M.D.; Harold N. Moyer, M.D. Volume IX, Physiology, Pathology, Bacteriology, Anatomy, Dictionary. Edited by W. A. Evans, M.S., M.D.; Adolph Gehrmann, M.D.; William Healy, A.B., M.D. Volume X, Skin and Venereal Diseases, Nervous and Mental Diseases. Edited by W. L. Baum, M.D., and Hugh T. Patrick, M.D. The Year Book Publishers, Chicago.

The sub-titles of these volumes indicate with sufficient clearness the field covered, and the names of their editors vouch for the value of the matter offered. They make an excellent and suggestive record of medical progress, and the arrangement and presentation are admirable.

RAILWAY AND OTHER ACCIDENTS WITH RELATION TO INJURY AND DISEASE OF THE NERVOUS SYSTEM. By Allan McLane Hamilton, M.D., F.R.S.E., Late Clinical Professor of Mental Disease in Cornell Medical College. William Wood and Co., New York.

With modern methods of flying through space so highly specialized, the need of works of this kind is obvious. Barring the excellent treatise by Bailey, no general work on this special subject has of late years been offered to the professional public. The present work is very commendable.

We cannot call it a treatise which is thoroughly modern, nor do we follow the author throughout in his point of view, but as the expression of personal experience it is a valuable addition to the store of knowledge on this subject. In some parts we feel that we cannot subscribe to all the author's statements. Thus we believe that it is bad policy to admit a traumatic element as directly causative of paresis. Still, the author's point of view is conservative in the main. As for locomotor ataxia, the opening sentence is very far from being clear. It concerns itself largely with a distinction without a difference, especially in these days when locomotor ataxia and tabes dorsalis are synonymous terms.

The chapter on spinal cord injuries is by no means complete, the symptomatology in particular being very fragmentary. Peripheral nerve injuries are treated at length and this chapter is very useful. The most interesting part of the book concerns the question of the legal aspects, and on this account the chapters on fraud and prognosis in relation to the verdict will be read with considerable interest. GOODALE.

TEXT-BOOK OF INSANITY. Based on Clinical Observations, for Practitioners and Students of Medicine. By Dr. R. von Krafft-Ebing, Late Professor of Psychiatry and Nervous Diseases in the University of Vienna. Authorized Translation from the last German Edition, by Charles Gilbert Chaddock, M.D., Professor of Diseases of the Nervous System in the Marion Sims-Beaumont College of Medicine, Medical Department of St. Louis University, St. Louis, Mo. With an Introduction by Frederick Peterson, M.D., President of the New York State Commission in Lunacy. F. A. Davis Company, Philadelphia.

Probably to the average doctor the name of Krafft-Ebing is somewhat unsavory, since it has been associated with the book "Psychopathia Sexualis." That work was a masterly study of some low forms of life that encumber the nets of a specialist. It was, doubtless, the fault of publishers and book agents that it got into the hands of many who could see in it only the slime adhering to the scientist's specimens.

This text-book of insanity is a different thing—the ***magnum opus*** of a great specialist in mental disease. The first edition appeared in 1879; the last in 1903.

The period before 1879 was one of analysis—of psychological dissection. Krafft-Ebing studied minutely the mental processes in his patients and classified them accordingly. This led him to recognize under the term "combined psychoses" (p. 203, translation) those remarkable cases, by no means rare, in which one form of insanity complicates another; paresis, for example, supervening in epilepsy. It also led him to describe nine types of epileptic insanity and ten forms of paranoia.

The period of synthesis was beginning in the work of Kahlbaum on Hebephrenia and Katatonia, but Krafft-Ebing's mission was not for it. The 1903 German edition contains (pp. 370-377) a fair account of Dementia Præcox in its three forms; but this is like a foreign body in Krafft-Ebing's book; there is no shifting of his views on melancholia and mania to make room, as Kraepelin does, for the newer conception. The translation contains nothing on Dementia Præcox.

This text-book is an anatomy of insanity—a fine dissection of the subject—especially valuable to the reader who has had narrow clinical opportunities, or who is in the dissecting stage of his own development. The encyclopedic descriptions of mental states associated with bodily diseases (pp. 167-198) are scarcely equalled elsewhere, even the polyneuritic (Korsakoff's) psychosis being included (contrary to the statement of a reviewer in the *Journal of the American Medical Association*), although the translator has unaccountably abridged the section (p. 178 in the English, p. 179 in the German) on this disease, so that the most interesting symptom ("fabrication") is not mentioned.

But much of psychiatry has undergone re-solution and re-combination; new forms, such as dementia præcox, have crystallized out of it; certain old forms, like mania and melancholia, have been made smaller and more clean-cut; many symptomatic states have fused, under the heads of delirium, confusion and stupor; and Krafft-Ebing's propensity for psychologic explanation is not in favor. For Kraepelin is king now, as Krafft-Ebing was twenty years ago.

The translator, Dr. Chaddock, has done his work well in most respects. He has a few affectations—"statistic" and "phthisic" and "typic"; in places he wants precision, as in "the delusion of the secondary maniac" (p. 353) for der Wahn des Verrückten;" and on the same page, "*evidences*" for "*recrudescences* of the primary stage."

It is only justice to say, however, that the translation is really excellent, and that it was needed. WILLIAM PICKETT.

TRAITÉ DE PATHOLOGIE MENTALE. Publié sous la direction de M. GILBERT BALLET, Professeur agrégé a la Faculté de médecine de Paris. By Messieurs. D. ANGLADE, F. L. ARNAUD, H. COLIN, E. DUPRÉ, A. DUTIL, J. ROUBINOVITCH, J. SEGLAS and CH. VALLON. Octave Doin, Paris.

This is one of the most extensive of recent French treatises on Psychiatry. It contains a systematic treatment of the subject of diseases of the mind in a large volume of 1,600 pages. It may be termed a series of monographs by the collaborators of the work, woven into a united volume.

It is, however, more than a mere text-book on Insanity or Psychiatry. These are usually the outcome of the experiences with types of mental phenomena which are encountered within the walls of asylums, but Ballet would view the subject in the larger light of general mental pathology, rather than in the restricted sense of the disorders of the mind that determine social irresponsibility. Thus, while it is held that no doubt the most important of the mental disorders are those that may best be observed in the asylum wards, yet there are a large variety of others that are not ordinarily met with in such practice. Thus delirium, as a pathological process, is of interest from this point of view, whether it occur in mania, in typhoid fever, in chronic hashisch poisoning or in erysipelas. The aboulias of the hysterical, the phobias of the neurasthenic demand explanation from the same standpoint.

Classification is held by the author to be a type of necessary evil. Defective they all must be in view of the many gaps of knowledge concerning etiology, pathology. symptomatology, etc., the classification, therefore, is considered more as a table of contents, roughly arranged in groups so that similarities and contrasts may most effectually be pointed out.

The work in general is divided into nine sections or books. Ballet, in the first section, begins with a short historical resumé of the development of the general subject of general psychiatry. The steps are well traced and the various strata of development carefully and picturesquely unfolded.

General Etiology, by D. Anglade, is a most exhaustive presentation of this subject. It is a very thorough and highly practical chapter. This is followed by the chapters on the General Diagnosis of Mental Affections and the Symptomatology, by J. Seglas. The latter chapter is a book in itself. Two-hundred pages are devoted to the elaboration of this portion. It is a most minute and valuable presentation.

Book II. is devoted to a consideration of the Syndromes Mania and Melancholia. The standpoint adopted being that they not only have no exact picture that may be regarded as typical, nor do they have a single cause. Thus mental confusion is a symptom not a disease; it may have its origin in various causes; as also may acute delirium. In either case the etiological factors are often so obscure that it is better, lacking such knowledge, to consider these affections as clinical entities. Mania has been written by D. Anglade and Melancholia by D. Anglade and G. Ballet.

In Book III. the mental affections, due to the *infections* and *intoxications*, are considered by various authors. J. Roubinovitch takes up the General Considerations, also the Febrile Deliria, Septicemic Deliria and the Delirium of Collapse, and. the External Toxic Psychoses, Alcoholic

Phychoses being well developed. D. Anglade writes on Acute Delirium, Mental Confusion (Meynert's Amentia).

Book IV. deals with the Constitutional Psychoses, under which head are included the Primary Systematized Deliria, acute and chronic; Periodic and Circular Insanity, both held to depend on a latent predisposition; while classified as a result of apparent predispositions are the phobias, obsessions, impulsions, moral insanities and deliria of persecution, etc.

Book V. considers the neuroses from the standpoint of the mental signs. Hysteria, neurasthenia, epilepsy and chorea are here dealt with. The organic psychopathies are treated of in a separate section. General Paresis and the Encephalopathies of acute and chronic character comprise the sixth book. Idiocy and imbecility in a seventh. Thyroid Insanities in another. The Treatment, both medical and administrative, makes the closing chapters of this valuable treatise.

This summary of contents does not in any sense convey an idea of the great helpfulness of this volume to the student and the specialist alike. It is a masterly work and deserving of a wide recognition.

Jelliffe.

Electro-Diagnosis and Electro-Therapeutics. By Dr. Toby Cohn, Nerve Specialist of Berlin. Translated from the Second German Edition and Edited by Francis A. Scratchley, M.D., Chief of Clinic, University and Bellevue Medical School. Funk & Wagnalls Company, New York and London.

Cohn's work has been before the medical public for a number of years, and in its original German dress has proven of great service to neurologists and general practitioners alike.

In its translation into English it will find more friends. It will aid the cause of electro-therapeutics, which has fallen very much from grace, because of the over enthusiasm of many practisers of this type of therapeutics, who have been unable to dissociate electrotherapy from psychotherapy.

For the physician who would seek a concise and practical guide as to the methods of electrical diagnosis we know of no better work. The charts of the electrical points of stimulation are useful and well reproduced.

As for the chapters on therapeutics they represent the most conservative and rational practise of one who has had a large experience. The practitioner who would seek in treatment all that the author claims will probably not be disappoined. This much cannot be said for many treatises on electro-therapeutics.

The translation is facile and the general book making is all that could be desired. Goodale.

Die Färbetechnik für das Nervensystem. Von Dr. Bernhard Pollack, Augenarzt in Berlin, pp. 158, Dritte, wesentlich erweiterte Auflage. Berlin, Verlag von S. Karger. 1905.

Two previous editions of this book have appeared, the first in the early part of 1897. The object of its publication is to give to those interested in the microscopic examination of the nervous system such directions as may be useful in their work. It is not intended for beginners in histology, but merely to supply such information regarding technique as the special student of the nervous system requires. Judgment has been used in the selection of methods, but in spite of this fact the present edition of the book, with its index, requires 158 octavo pages. After a considerable interval this new edition appears, necessitated by the advance which has been made in histological technique during the intervening period. The development of neuroglia methods and the methods of Bethe and others for neuro-fibrils have necessitated a certain enlargement of the work. In addition to the technique of staining methods, the cutting, hardening and preserving of the central nervous system are discussed at considerable length, and many useful suggestions made regarding work in this somewhat difficult and involved field. Particularly useful, though perhaps subject to differences of opinion, is a brief list of methods recommended in the demonstration of the separate portions of the nervous system. In spite of Mallory's notable contributions to the staining methods of the nervous system, but one of his is given space. His neuroglia methods, for example, are not noticed. In general, the book is well arranged, easy of reference, and should be a most useful guide. E. W. TAYLOR.

News and Notes

To Members of the American Neurological Association: The Council announces that the thirty-first Annual Meeting will be held in Philadelphia on Thursday, Friday and Saturday, June 1st, 2d and 3d. Therre will be two sessions daily, from 10 a.m. to 1 p.m. and from 2:20 to 5 p.m.

If it is your intention to contribute a paper, your attention is called to a clause of Article VII of the Constitution, which states: "The reader of a paper shall not exceed twenty minutes in the presentation of his paper, and no one shall speak longer than five minutes in the discussion of a paper."

It is the earnest request of the Council that you will, if practicable, present a verbal abstract of your paper instead of reading it in full.

Your attention is called to another clause of Article VII, which states: "Members must send the titles and *abstracts* of their papers to the Secretary at least six weeks before the Annual Meeting." Titles and abstracts must, therefore, be received by the Secretary on or before April 20th.

The Council recommends the following candidates for election to active membership: Dr. Wm. G. Spratling of Sonyea, Dr. L. Pierce Clark of New York, Dr. F. H. Weisenburg of Philadelphia, Dr. Alfred Gordon of Philadelphia, Dr. William Pickett of Philadelphia, Dr. Charles S. Potts of Philadelphia. The annual dinner will be held on Friday evening, June 2. The Council announces that the annual dues for 1905 will be five dollars. Very respectfully yours,

Graeme M. Hammond, Sec'y.

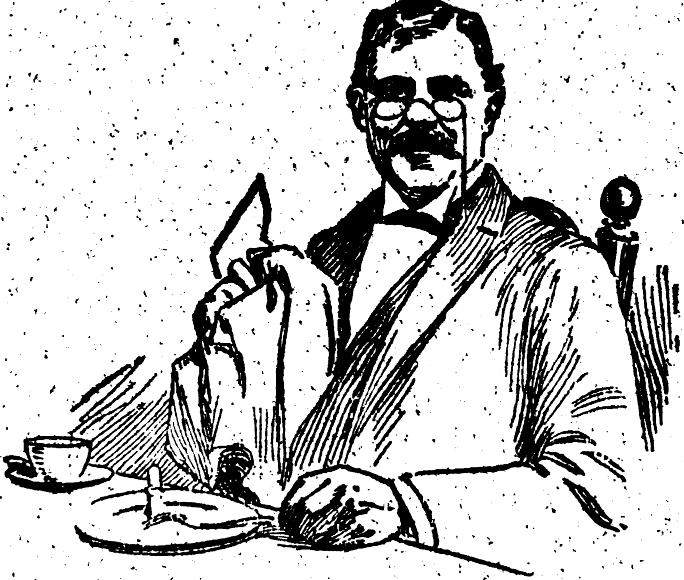
The National Association for the Study of Epilepsy and the Cure and Treatment of Epileptics.—There is a widespread belief that the Transactions of the Association at the reecnt meeting should be printed. The very creditable publication, showing the transactions of the Association at the meeting held in Washington in 1901, has set a standard which it is hard to equal. I have the opinion of the President of the Association and some of the members of the Executive Committee that we should try to issue a volume of about the same size and general appearance, but that we may, if needs require, omit the illustrations.

We would attempt to include an outline of the proceedings at the meetings of 1902 and 1903.

The cost of publication will be near $400. Towards the amount the following subscriptions have already been offered: Mr. Letchworth, $25; Dr. Spratling, $25; Dr. Bullard, $25; Dr. Flood, $10.

Your coöperation is needed. Will you kindly contribute as liberally as possible? You will be entitled to one copy of the "Transactions" for every dollar you contribute; but it is hoped that you will leave all but one copy in the hands of the Secretary and instruct him to use such proceeds as he may realize from them for the needs of the Association.

Everett Flood, Sec. and Treas.



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THE

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Original Articles

ARTERIOSCLEROSIS IN ITS RELATION TO DISEASES OF THE NERVOUS SYSTEM.

BY E. D. FISHER, M.D., AND HARLOW BROOKS, M.D.,
OF NEW YORK.

CLINICAL ASPECT. BY EDWARD D. FISHER, M.D.

The changes in the arteries, as far as the pathological condition can be demonstrated, show that arteritis or arteriosclerosis, whether due to infection or so-called senile changes is the same in character, at least in the later stages. Still the clinical picture is so different, as a rule, that one is forced to come to the conclusion that later studies under improved methods must demonstrate that we have to do with distinct lesions, at least in the early stages, whatever the end results may be.

Even from what has been demonstrated we can assume that syphilis has a tendency to cause a periarteritis, involving, perhaps, the adventitia or external coat primarily, and leading later to great thickening of all the coats, which thus occludes the lumen of the vessels, causing thrombosis.

While, indeed, we observe clinically that cerebral hemorrhage occurs in syphilitic endarteritis, it is far less frequent than thrombosis. Atheroma, on the other hand, usually primarily involves the intima, ultimately affecting the other coats, and, by subsequent degeneration, forms patches of calcification which may be continuous in the long axis of the vessels, without marked thickening of the coats of the vessels.

This arteriosclerosis is very general, involving the smaller arteries and leading to hemorrhage through small miliary aneurisms of the small straight arteries in the brain.

Studying the various diseases of the nervous system as resulting from arteriosclerosis, whether atheromatous in type or of syphilitic origin, the most frequently observed are apoplexy, whether due to occlusion of the vessels or hemorrhage; general paralysis of the insane: multiple sclerosis: cerebral atrophy, and cerebral syphilis, which must be recognized as a distinct entity.

We will first consider cerebral apoplexy, the one most frequently observed. One finds that hemorrhage into the brain is most common after forty, while it may occur in children following the infectious disease in which an arteritis (acute) is present: or, again, cases of marasmus have been reported, with definite pathological changes, as advanced arteriosclerosis, probably chronic in its course. The usual clinical picture, however, in the first class of adult cases of hemorrhage, is marked arterial degeneration as demonstrated during life by the hard, thickened vessels observed especially in the radial and temporal arteries, and which can be observed also in the retinal vessels, and while not clinically demonstrable is also present, as shown by Broadbent, in the aorta. There is usually high arterial tension and the arteries are incompressible. We early have cardiac hypertrophy, which later passes on to dilatation; associated with this there is often evidence of interstitial nephritis, as shown by low specific gravity of the urine; intermittent or constant albuminuria and casts; and not infrequently the ophthalmoscope demonstrates retinal hemorrhage. The only etiological factors we may find in these cases may be overwork or strain—physical or mental; overeating; moderate or excessive use of alcohol: gouty, or less often rheumatic diathesis; or heredity.

The course of a case such as this may be short or protracted, frequently modified by change in the method of living or medicine. It is impossible in these cases to state whether the nephritis is an important factor in causing the disease. No doubt in some it is, the defective elimination of the toxins by the kidneys causing the high tension in the arteries, and acting especially in causing the arteritis of the small peripheral vessels, by which they lose their elasticity and offer much resistance to the course of the blood, then, again, secondarily calling upon the heart for extra exertion and causing its hypertrophy as well as hypertrophy of the muscular coats of the vessels themselves, the larger as well as the smaller, for we must not forget that in the circulation we have two important elements in its propulsion, the heart itself and the muscular elements of the arteries, sometimes called "the second heart."

However, while we must recognize nephritis as a cause of arteriosclerosis, still, as Broadbent has said, many cases are due to a primary arteriosclerosis, which has involved the arteries of the whole body, including the kidneys, so that we find disease of the cerebral vessels, of the aorta, of the coronary arteries, the vasa vasorum of the arteries themselves, and of the various organs, kidneys, liver, and the peripheral arteries.

Another class of cases, however, which also result in hemorrhage, do not present the evidence of high tension or thickening in the walls of the vessels; in fact, post mortem the arteries at the base appear thin and soft. In these cases, however, the arterioles are found diseased in the region of the internal capsules, and liable to rupture.

Perhaps at this time it would be well to refer to the cases of cerebral hemorrhage occurring in typhoid fever, scarlet fever, and the other infectious diseases, or, again, in rabies and in the puerperal period. We are dealing here with probably an acute arteritis as shown in the case of rabies related by Dr. Brooks, with the added change in the condition of the blood which renders the blood less capable of being transmitted through the blood vessels. Here we commonly have hemorrhage, or less frequently thrombosis. The latter condition of thrombosis was seen in a young woman, aged twenty-two, a primipara, who, in the third week after confinement, had hemiplegia in the right side, associated with which was the usual mental disturbance found in puerperal mania. As the patient completely recovered from the paralysis and aphasia, although not demonstrable by autopsy, I can only consider that we were dealing with a thrombosis due to blood changes, which was ultimately carried away in the circulation or absorbed.

Different, indeed, is the condition of syphilitic hemiplegia. In a typical case occurring in early life, usually six to ten years after the primary lesion of syphilis, there is no evidence of high pulse tension, no cardiac hypertrophy or nephritis, the peripheral arteries show no signs of hard, resisting walls. While the onset may be that of a hemorrhage, the usual condition is due to thrombosis. The onset is usually slow; the patient may, indeed, at no time lose consciousness, and may be able to fully describe the course of the onset and the subsequent complete hemiplegia and aphasia.

The commonest situation of cerebral hemorrhage is in the region of the internal capsule. While we find the arteries at the base of the brain in the circle of Willis the seat of atheromatous changes, as well as the basilar artery, they rarely are the seat of hemorrhage. The reason why the smaller internal vessels are usually affected is that they enter the brain perpendicularly, are straight in their course, and do not anastomose. In atheroma of the vessels involving the large arteries, there is no interruption to the full impact of the blood and no distribution as in the circle of Willis, and their diseased walls soon give way.

Thrombosis and aneurism, on the other hand, are most commonly found in the pons and at the base.

Apart from the disease just considered, perhaps the most usual cerebral disease is general paresis, or dementia paralytica. This is essentially a vascular disease, and in the majority, if not in all cases, due to syphilitic endarteritis, if we can use such a term, which, as I have previously said, recent pathology seems to deny.

Adolf Meyer, in a recent communication in regard to the arterial changes found in general paresis, says that there is nothing to distinguish them from the usual form of arterial disease in which there is no mental disturbance. Arteriosclerosis of the heart and the aorta, and nephritis, are often found in the insane; but it is not often that insanity can be directly referred to them.

In general paresis we find a chronic progressive meningo-encephalitis, associated with cranial nerve lesion and degeneration in the spinal cord tracts, either of the posterior columns or lateral motor tracts, dependent on arteriosclerosis.

We are evidently not dealing with a direct syphilitic infiltration in this disease, but the tendency to arteriofibrosis which syphilis impresses on the system as a whole. The same can be said of tabes. We do not find the vessels the seat of gummatous infiltration, as in cerebro-spinal syphilis, but, rather, subject to changes characterized by general thickening of the walls, thus interfering with the general nutrition.

While some writers have drawn a very close analogy between general paresis and tabes, reporting cases of the latter passing into general paresis, I have rarely observed a typical case of tabes become paretic. We have in both diseases the Argyll-Robertson pupil peculiar to both of these diseases, and these only: but a patient with marked ataxia, diplopia, optic atrophy, lightning pain, etc., may thus continue for years without change or even with an improvement in his symptoms without at any time showing the slightest mental disturbance.

Few cases of general paresis occur without evidence of some spinal involvement, and especially in the class of atypical cases which last for seven or eight years. Here there is often marked sclerosis of the motor tracts of the cord, or, again, of the posterior tracts. Post mortem we usually find degeneration in both tracts, but rarely so definitely confined to one system tract as in tabes. On the other hand, in old cases of tabes, it is not unusual to find the lateral tracts also to some degree affected.

While in tabes we cannot hope for a cure, all such reported cases being to my mind cases of mistaken diagnosis, being either spinal syphilis or multiple neuritis, still much can be done to alleviate symptoms. The ataxia, by re-educating the muscle, can be reduced; and, again, one must not forget that the disease itself often comes to a standstill, either permanently or for a time. I encourage all tabetics to do anything they have the power or strength to do, especially as no mental or physical exercise can at all injure them or hasten the course of the disease.

Multiple sclerosis is due to arteriosclerosis. It is a comparatively rare disease, occurring in about one in two hundred cases in thirty-five thousand patients, presenting themselves at a clinic for nervous diseases.

There is much difference of opinion as to whether syphilitic endarteritis, to still use that term, is ever present.

The pathological changes observed in the vessels are increase in the volume or thickness of the intima, with narrowing of the lumen, or, indeed, obliteration, although this is not always found.

It is thus an inflammatory disease commencing in the blood vessels. One finds also the lymph tracts affected, and dilatation of the perivascular lymph spaces.

Erb, Gowers, Strümpell, Oppenheim and Rumpf do not consider syphilis as a factor in the disease, while Westphal and Fournier are less positive. Leyden and Goldscheider have written of the difficulty at times of differential diagnosis between spinal syphilis and sclerosis (multiple).

Buckholz reports a case which falls under the head of multiple sclerosis. In the necropsy, however, there were many syphilitic evidences; i.e., of the testicles, gumma of the temporal lobe, and specific disease of the vessels and of the pia mater. Bechterew called his case syphilitic disseminated cerebrospinal sclerosis. Schulze's case partly resembled general paresis and was distinctly specific. Rosenfeld wrote an article on endarteritis and multiple sclerosis (*Archiv für Psychiatrie und Nervenkrankheiten,* Vol. 38, No. 2, 1904). His case was typical in its clinical history. The necropsy showed the usual sclerosed spots in the brain, pons and cord; the axis cylinders persisted at least in part; some vessels showed hyaline degeneration without occlusion. The basal arteries showed disease of the intima and adventitia. In some arteries occlusion was present.

The writer does not say, however, that this is characteristic of syphilitic changes, nor does he give what is the differential characteristic.

Arteriosclerosis may be the cause of diffuse inflammation of the spinal cord; or, rather, we may define it as a combined disease of the various tracts, giving, therefore, symptoms resembling tabes and spastic paralysis. We find in the cord, as in the brain, arteriosclerosis, atheroma of the vessels, hemorrhage and thrombosis. This accounts for irregular cases of paralysis occurring in the old.

Syphilis of the brain characterizes itself by headache, mental dulness, tendency to coma, cranial nerve paralyses as seen in diplopia, ptosis, irregularity of the pupils with slowness of reaction, often transient or permanent paralysis; and, again, mental disturbances of excitement and peculiarity of speech or utterance which at times make a differential diagnosis from general paresis very difficult.

This applies also to spinal syphilis when the lesion may be so situated as to involve the posterior columns and give many if not all the symptoms of tabes. The diagnosis can be made, however, by the rapidity of the onset of the symptoms, which is rare in tabes, and also by their rapid disappearance.

We are dealing in these cases with a gummatous infiltration and not a true arteriosclerosis, which is capable of absorption under specific treatment.

Arteriosclerosis seems to me to be the basis of angina pectoris. While all our autopsies do not show disease of the coronary arteries, still general disease of the arterial system is often present in these cases. Syphilis cannot be excluded as a causative factor here.

In all cases of arteriosclerosis we must look to the predisposing cause. The kidney and heart should be carefully examined, and any deficiency of action compensated for. Overwork, overeating, sedentary habits, and the use of alcohol in excess must be modified. If a specific history, large doses of the iodides, especially in the early stages of the disease are required.

Again, I have found, when the pulse is of high tension, that thyroid extract, as, from its physiological action, one might expect, is beneficial.

In some old cases of atheroma I advise small doses of the iodide of potassium; i.e., five to ten grains, and thyroid extract, five grains, two to three times a day.

We must not forget that many of our syphilitic cases have other changes in the arteries also, due to alcoholic excesses or overwork, etc., and we may have in them also high arterial tension, nephritis and cardiac hypertrophy. In these cases much relief is obtained from continued dosage of digitalis and nitro-glycerine combined, in conjunction with some form of iron, either Blaud's pills or the newer combinations, as ovoferrin, etc. In these cases the old calomel purge once a week is not to be forgotten. I usually advise it in small doses, either one grain, or one-tenth frequently repeated. A drug also which is variously estimated as to its value, which I have long used is the proto-iodide of mercury in 1-6 grain doses, t.i.d., and this I use whether the case is syphilitic or not.

Brunton advises, for high blood pressure,

Potassium bicarbonate	grs. XXVIII
Potassium nitrate	grs. XVIII
Sodium nitrite	grs. IVss

in a glass of water in the morning.

Traumek's saline solution (inorganic serum) hypodermic,

Sodium sulphate	grms. 14
Sodium chloride	grms. 4.9
Sodium phosphate	grms. 15
Sodium carbonate	grms. 21
Potassium sulphate	grms. 40
Aq. Dist. ad	grms. 100

Dose, 1 c.c. every four to seven days.

—*Med. Press*, Nov. 26, 1903.

SUMMARY OF THE PATHOLOGY OF CEREBRO-SPINAL ARTERIOSCLEROSIS.

BY HARLOW BROOKS, M.D.

Arteriosclerosis of the brain and spinal cord is occasionally found as a purely local disease and it may even be seen alone in either the brain or cord. It is, however, usually found associated with more or less general arterial disease, as a local manifestation more pronounced in the cerebrospinal tissues than elsewhere. There are reasons why this is so frequently the case, but in considering them we must also recall that the brain and cord are not alone in this peculiarity since a local arteriosclerosis of the kidney, of the pancreas and of any of the other special viscera is by no means infrequent.

Coplin finds, according to his statistics, that the cerebral arteries are more prone to arteriosclerosis than those of any other viscera, placing them as fourth in the frequency of involvement. (1, arch of aorta, 2, thoracic and abdominal aorta, 3, iliac arteries, 4, cerebral arteries.)

The reasons for this preponderance of local arteriosclerosis are variously stated. It is not due to any inherent structural peculiarities in the arteries themselves though the relation of the trunks to the surrounding tissues, and the nature of these is an important factor. Thus in the brain and cord the vessels are not united, as in most other parts of the body, directly to firm and resistant tissues, but are, in large part suspended in channels, the perivascular lymph spaces, which are crossed over by but a fragile frame-work of connective tissue and this joins the vessels to the soft, semi-gelatinous nervous pulp. This arrangement renders the vessels particularly susceptible to the mechanical factors productive of cerebro-spinal arteriosclerosis. Added to these peculiarities of structure is the fact that many of the cerebral arteries are "straight arteries," that is their axis is in a direct line of the blood stream, and on that account they are particularly subjected not only to the highest mean blood pressure, but also to the direct blow of the cardiac systole. As is well known these same arteries are prone to the lodgment of bacterial emboli, which are among the unquestionable excitors in many instances of arteriosclerosis. Dana and others have shown that in some conditions cerebral atrophy, by a lowering of the external or supporting pressure on the cerebral arteries favors over-distension and the origin of inflammatory processes. Still other inductive factors are at work, such as for example the great physiological activity of these trunks, but the chief points have already been stated.

The causes of arteriosclerosis of the central nervous system do not differ from those producing the lesions in the general arterial tree, and we may primarily classify them under physiological and pathological.

Physiological arteriosclerosis is a normal process in old age. The anatomical changes characteristic of senile or physiological arteriosclerosis do not differ materially from the changes seen in the pathological condition. Minor differences in the location and extent of the lesion and in certain special and inconsequential characteristics, exist in some instances but generally to only a negligible degree. In old age the condition is found as among the early signs of malnutrition and beginning tissue death.

Pathological arteriosclerosis may be broadly and yet not inaccurately defined as premature or abnormally induced arterial senility. The etiological factors may be best considered under the headings of mechanical, infectious and toxic. In many cases all are concerned, and in most, more than a single agent must be considered. Some observers also cite heredity as one of the determining agents, and doubtless the disease is hereditary in some instances, but only where the transmission of formation of certain toxic substances takes place or where exposure to certain conditions predisposing to the disease exists in families from generation to generation. We must admit, however, on theoretical grounds, the possibility of purely fetal characteristics which may predispose, as in maldevelopment of the vascular system, or some inherent flaw, such as for instance probably exists in hemophilia.

Under the mechanical factors must be considered all those conditions which induce abnormally high blood pressure, such as physical overwork, particularly exemplified in improperly trained athletes, or where from any reason the blood pressure is relatively raised, as for example in caisson workers. It is notable that it is much more commonly caused where the blood pressure is raised to a high point sporadically, as in athletic contests, and relatively less often where the pressure is habitually raised, as in laborers.

Infectious agents usually act through the formation of bacterial emboli or thrombi, from which infection of the vessel walls extends. External infections through the adventitia may also occur in cases, as where a vessel passes through an inflammatory area. Stengel states that periarteritis nodosa is usually caused in this manner, though also admitting that it may at times have a definite special pathology.

Toxins are doubtless the more common and important etiological factors concerned in arteriosclerosis. Certain of the toxemias are of infectious origin, as for instance in diphtheria or chronic tuberculosis. In other instances the poisons may be autogenous, as in habitual constipation, over-alimentation, or in malnutrition of various types. The more common toxic conditions are those occuring in alcoholism, chronic nephritis, lead poisoning, gout and rheumatism. In nearly all these conditions it is practically certain that the causative factor is not a simple one, but that in each case it is complicated by other secondary agents: thus excessive blood pressure takes place in nephritis, and in nearly all the conditions, intestinal toxemias, malnutrition and other subsidiary factors are present.

Syphilis is one of the most common disease conditions producing arteriosclerosis in its most exquisite types. Arteriosclerosis is almost invariably present in syphilis, and cases of this contagion in which arterial disease never develops, are the exception. Syphilitic arteriosclerosis is particularly prone to attack the vessels of the central nervous system. The reasons for this generally admitted fact are various; probably the most logical of them is the idea propounded, we believe by Dana, namely, that the syphilitic poison acts especially on the cerebral arteries, since they are literally bathed on both sides by the blood and toxic lymph circulating in the perivascular lymph spaces.

Numerous types of arteriosclerosis are differentiated by various authors, and unfortunately hardly any two agree absolutely on even the simple definition of arteriosclerosis. The disease is sometimes classified, according as it is chiefly manifested by alterations in the intima, media or adventitia, but this is very unsatisfactory, since one rarely or never finds a case in which all the arteries of one body or even viscus are changed in precisely the same manner. It is also rare to find an artery in any except the acute stages in which the process is limited solely to any one coat; hence it is that these finer classifications are inaccurate anatomically, and it is well for us to realize that except for acute or specific types of arteriosclerosis, such as septic or tubercular, the condition is a general one differing only in degree or in the type of the secondary, i.e., degenerative alterations, which follow the primary inflammatory manifestations.

Some authors classify arteriosclerosis anatomically according to the cause of its development; they thus speak of alcoholic arteriosclerosis, syphilitic arteriosclerosis, and so on. Now as a matter of anatomical fact, these distinctions do not exist. Syphilitic arteriosclerosis, the type most minutely differentiated, usually does not differ anatomically from many other forms which may be caused by other factors, except where the walls of the artery are actually the seat of a gummatous or syphilitic process. With this statement we must, however, admit that certain forms are more frequent where syphilis is present than in non-syphilitic cases, thus periarteritis is more common in lues, probably for the reasons already cited, and fatty degeneration is also more often seen than is calcareous infiltration of the arterial walls.

It remains then for us to classify the various forms of arteriosclerosis as acute or chronic, remembering that these merge one into the other with no distinct line of demarcation. The condition may also be quite correctly classified according to the type of alteration or degeneration predominating in the diseased trunks.

Concerning the manner of development of the changes in arteriosclerosis much experimentation and many observations have been made so that now, though many points still remain unknown the more common methods of formation are fairly well demonstrated, particularly by the work of Gall and Sutton, of Thoma, Koster and others. The older pathologists thought that as a rule the fibrous hyperplasia, which is characteristic in greater or less degree of all forms of the disease, was immediately excited by the direct irritation produced by some abnormal substance circulating in the blood, so acting on the intima primarily and secondarily perhaps on the remaining arterial walls. Of course, where the arteries are surrounded by an inflammatory area, as when they pass through an abscess, direct extension from the adventitia is admitted.

Although this theory no longer holds first place in the minds of most students, it is certainly one of the methods, as has been amply demonstrated by experiments. Undoubtedly the chief condition underlying the development of the disease lies in any state which decreases the elasticity of the arterial wall, whether permitting it to over-distend or preventing its physiological contraction. This may be brought about by many different changes in the walls of the artery. In those cases in which the disease seems to be inaugurated as a result of excessive blood pressure, it is probably brought about by over-distension, resulting in injury of the fenestrated layers and possibly of the muscle coat, with the result that the lumen of the artery remains more or less permanently distended. Probably as an effort on the part of nature to restore the normal caliber of the tube, and, at the same time, to strengthen the weakened and dilated walls, hyperplasia of the areolar connective tissues takes place. Only the white fibrous elements take part in this hyperplasia; the yellow elastic fibers do not hypertrophy, whereas in physiological thickening of the arterial wall such as takes place in the arteries of well trained athletes or in the vessels of laborers hyperplasia of this coat does eventuate.

In certain cases this hyperplasia, which may be compared to like processes taking place in numerous similar conditions in other parts of the body, becomes so marked as to limit the lumen of the artery to a greater or less extent. At the same time the strength of the wall is decreased rather than increased, not only on account of the lack of elasticity of the tissue formed, but also since it is of low grade and is very likely to undergo various types of degeneration, notably fatty and calcareous. Degeneration takes place whether blood vessels are formed in the new tissue or not, though sometimes trunks springing from the vasa vasorum are very numerous and extend into the innermost coat of the intima.

This sequence of affairs is induced not only when the integrity of the arterial wall has been impaired by increased blood pressure or direct traumatism, but also when, through any cause whatsoever, the physiological activities of the arterial walls are obtunded. It therefore occasionally occurs as the result of trophic faults following disease of the nerve fibrils which normally control the media or muscle coat. One frequently finds extreme examples of this variety in the vessels supplied to paralyzed extremities. It is stated that in such instances the interstitial changes are most pronounced in the media coats, but unless seen in the early stages this is difficult to demonstrate, since it very soon also extends to the other walls of the artery, which may have become diseased as a secondary process, following the incompetence of the muscle coat.

Probably the most frequent factor back of these arterial changes lies in conditions which impair the nutrition of the arterial walls. One of the most frequent of these is a diseased condition of the vasa vasorum, on which the arterial walls are immediately dependent for their nourishment. If the caliber of these minute vessels becomes obliterated or limited by any process, such as arteritis or thrombosis, the portions of the arterial walls depending on these particular vessels for their nourishment lose their elasticity, the power of contraction or relaxation, and the vessel at this point distends under the blood pressure, and the hyperplastic alterations mentioned become set up as an attempt at compensation.

Another type of arterial disease depending for its origin on the vasa vasorum is seen in those cases of arteriosclerosis where the nutrient arteries are surrounded by exudates of small round cells. Arteriosclerosis is undoubtedly inaugurated in this manner by a primary arteritis of the vasa vasorum accompanied by cellular exudate from which the process becomes diffused. The "periarteritis simplex" described by Charcot, which occurs frequently in the cerebral arteries and leads to the formation of miliary aneurisms, is probably of this manner of formation. The disease is then primarily in the adventitia, but subsequently spreads to the other coats, the degeneration of which produces the deficient areas which subsequently become dilated into aneurisms of more or less size.

Another disease condition probably sometimes inducing arteriosclerosis through the weakening of the arterial wall, is where some type of primary degeneration, as hyaline or albuminoid, invades the coats which then dilate and is followed by interstitial hyperplasia. It is more probable, however, that the interstitial hyperplasia is generally succeeded rather than preceded by such forms of degeneration.

It is foreign to the subject of this paper to attempt a decription of the various anatomical varieties of arteriosclerosis, and at best this is a thankless task since the varieties described are so numerous. All can however be included under the headings of the various types of degeneration following acute, subacute and chronic inflammatory disease affecting the walls of the arteries. As already stated the type of the degenerative change is not usually found to be the same throughout the entire body, and the various classifications are more properly considered as stages in the same process than as different forms of a disease.

SYPHILIS OF CEREBRO-SPINAL AXIS.

Showing marked arteriosclerosis of the blood vessels of the spinal cord.

J. R., Montefiore Hospital. Aged fifty-three years. Widower. Occupation, manager. Family history negative.

Previous History.—Usual diseases of childhood. Has had gonorrhea "very often." Had a sore on penis 30 years ago, indurated and accompanied by enlargement of inguinal glands. Rash all over body followed, loss of hair of head and eyebrows. Was treated for syphilis for six months, symptoms all disappeared. Thinks that he had another hard chancre 27 years ago. Has had chancroids frequently, has had epididymitis and usual complications of gonorrhea. Patient states that he has had "Every disease that a lady can give a man." Has used alcohol and tobacco to excess. Masturbated as a young man, has constantly indulged in sexual excesses. Wife has had two miscarriages, both at about third month, no living children.

Present illness.—Twenty-two years ago had pains in toes with sensations of constriction in calves, finally typical girdle pains. Fell in street while running after a car, has not been able to run since, but could walk when assisted. One year later became cross-eyed and had diplopia. Became impotent. Lost control of bladder. Had respiratory crises, unsteadiness of hands, etc. Symptoms have increased up to present day.

Present complaints.—Excruciating boring pains all over. Inability to walk. Incontinence of urine. Ataxia in hands.

Physical examination.—Extreme ataxia in legs. Lower extremities very flaccid. Marked Romberg. Pupils do not react to light, not consensually, ocular movements impaired, convergent strabismus, nystagmus. No atrophies of upper extremities, though muscles are very flaccid, contractions, slow and vermiform. Reflexes present on both sides, but markedly ataxic. Knee-jerks, Achilles and plantar reflexes absent. Interscapular, epigastric, abdominal and cremasteric reflexes absent. Marked slowness of muscular contractions throughout. Abdomen and lower extremities anesthetic.

Examination of heart, lungs, liver, spleen and kidneys show nothing of especial note.

Appears to be mentally sound.

Present history.—After remaining in the hospital several years, showing very little improvement under the usual methods of treatment, patient suddenly died with the symptoms of pulmonary embolism.

Post-mortem examination showed: (Abstract of protocol.)

Heart.—Fatty degeneration and brown atrophy. Marked sclerosis of coronaries. Chronic endocarditis of aortic and mitral segments.

Aorta.—Marked endarteritis with extensive areas of fatty degeneration and occasional patches of calcification. Same present in large trunks of thorax and neck. Areas of necrotic softening in walls of arteries.

Lungs.—Thrombosis of both pulmonary arteries. Chronic pneumonia. Chronic bronchitis.

Liver.—Fatty degeneration with lobular cirrhosis.

Stomach and intestines.—Chronic congestion.

Spleen and lymph nodes.—Generally enlarged, hyperplastic lymph-adenitis.

Kidneys.—Numerous old infarctions. Chronic interstitial nephritis.

Brain.—Examination not permitted.

Spinal cord and medulla.—Irregular areas of both ascending and descending degeneration. Marked arteriosclerosis. (All findings verified microscopically.)

EXAMINATION OF ARTERIES OF SPINAL CORD.

Medium sized vessels.—The perivascular connective tissue throughout shows a moderate degree of thickening and a good many of the connective tissue cells in this distribution are filled with pigment. This change is also continued into the adventitia, but it is not very pronounced and no fibroblasts are found, the change being apparently a chronic one. The media coat shows very slight alteration in the vessels of this caliber. The muscle fibers are distinct, but are not separated by an abnormally abundant interstitium nor do these cells show any evidences of degeneration except in a few instances where slight hyaline changes are to be made out.

The internal fenestrated membrane is well developed in the greater number of the medium sized arteries, but is not abnormally thickened.

The intima coat is greatly increased, the thickness varies considerably, but in places equal three or four times that of the remainings walls of the corresponding vessels, though for the greater part the increase is not greater than two to three times the thickness of the normal intima. Fibroblasts and proliferating connecting tissue cells are abundantly present in this coat, so much so that in places the structure closely resembles that of embryonic connective tissue. Areas of fatty and atheromatous degeneration and ocasional small patches of round-celled infiltration are frequent. Hyaline degeneration is very pronounced in this coat in some of the arteries, while others are nearly free from it. Calcification is not present in any of the vessels of this class.

The endothelial coat is mostly intact, though over some of the more marked areas of thickening the cells show evidences of reproduction and are occasionally desquamated.

The vasa vasorum show very slight alterations and none which are constant.

As a rule the medium sized arteries are but moderately filled with blood.

Small sized arteries.—The supporting tissue of the small sized arteries and arterioles is perhaps slightly increased, but, it shows as its most marked characteristic hyaline degeneration extending out from the walls of the more extensively diseased arteries. The adventitia shows the same characteristics as the supporting tissue, but in the greater number of places remains practically normal.

The media and intima in most of the arterioles is fused into a single layer with a thickness about double that normal to these layers. The entire mass of the tissue shows a diffuse hyaline degeneration of very pronounced degree. Occasionally abnormal hyperplastic connective tissue cells can be made out, but as a general thing all structure except for an indistinct fibrillar mesh is obliterated.

The endothelium of the intima shows occasional large atypical cells, characterized by very large nuclei, irregular shape and small quantity of widely separated chromatin.

Many of the smaller vessels are enclosed in an abnormally dilated perivascular lymph space which is occasionally filled by granular detritus. Corpora amylacea are frequent about some of the vessels, but they appear to bear a more direct relationship to the connective tissue stroma than to the vessels as such.

Capillaries.—Unlike the arteries, the capillaries are, for the greater part, well filled with blood cells. A good many of them are surrounded by neuroglia which is perceptibly increased in density. Some of them show a partial blocking of the lumen with large atypical endothelial cells.

ARTERIOSCLEROSIS OF THE BRAIN AND SPINAL CORD, OCCURRING IN ALCOHOLISM.

J. M., male, aged forty-two years. Wd. 32 Bellevue Hospital.

Patient entered hospital in semiconscious state and not able to give a coherent history. Friends state that he has been a heavy drinker of whiskey and beer for years. He had delirium tremens several times. He has used alcohol constantly, but of late has been drinking more than ordinarily.

The physical examination and clinical course of the disease present the usual picture of delirium tremens, and patient died after four days in hospital.

Abstract of protocol. Verified by microscopical examination.

Heart.—Interstitial myocarditis, brown atrophy and fatty degeneration. Marked sclerosis of coronary arteries.

Lungs.—Chronic miliary tuberculosis of apices. Chronic congestion, emphysema and anthracosis.

Liver.—Fatty degeneration. Chronic congestion. Moderate interstitial hepatitis.

Spleen.—Chronic congestion with marked pigmentation. Periarteritis of smaller arteries.

Pancreas.—Chronic congestion. Parenchymatous degeneration.

Kidneys.—Marked parenchymatous with fatty degeneration. Acute congestion of blood vessels. Extensive epithelial desquamation. Moderate interstitial increase.

Stomach.—Chronic gastritis. Recent hemorrhages into mucosa.

Adrenals.—Extreme congestion with minute medullary hemorrhages. Fatty degeneration of parenchymatous cells.

Cerebrum.—Marked edema of pia-arachnoid. Old areas of meningeal thickening. Moderate cerebral edema and congestion, brain otherwise macroscopically normal. Microscopically a good many degenerated fibers are found in the subcortical layer of the Rolandic adeas. Certain ganglion cells of the corresponding gray matter show chromatolysis and atrophy.

Spinal cord.—Macroscopically normal. Microscopically a good many degenerated fibers are found, particularly in the direct and crossed pyramidal tracts and in the anterior radicular zone. There is a general increase in the connective tissue framework of the cord, and corpora amylacea are frequent in the interstitium adjoining the adventitia of many of the vessels.

Special examination of the blood vessels of the brain and cord.—There is a general dilatation of the perivascular lymph spaces in both the cord and cerebral tissues. Both small and medium sized arteries are similarly affected, but the dilatation is more marked about the smaller trunks. The supporting connective tissue of the arteries is generally increased, the spinal arteries showing this change in more marked degree than the cerebral vessels. The adventitia of most of the medium sized arteries is thickened in moderate degree; this is more apparent in the spinal vessels than in the cerebral. Very few hyperplastic connective tissue cells, that is fibroblasts, are demonstrable in this coat, but the process seems to have been a chronic productive one.

The media shows a production of connective tissue between the muscle cells, causing pressure atrophy, so that the coat is mostly replaced by fibrous connective tissue of the adult type. The disease of this layer is probably largely responsible for the abnormal distension of most of the vessels. The hyperplastic connective tissue is almost entirely of the areolar type and there appears to be little if any increase in the yellow elastic fibrils.

The subendothelial layer of the intima shows a more pronounced, though very irregular hyperplasia, and in this coat a few fibroblasts appear. Small patches of round cell infiltration are present, and in a few places where the thickening of the coat has been most pronounced, it is sufficient to cause bulging into the lumen, with disintegration and athermomatous degeneration of the hyperplastic tissue. As a rule little change is to be seen in the endothelial layer of the vessels of medium size, but a few of the cells show swelling. The endothelium of the vasa vasorum is seen to be considerably swollen in many places and many of the vessels are occluded with a clot, a part of which is of the white variety. Very little absolute alteration, aside from that mentioned, can be made out in the walls of these very minute vessels, but a considerable number of them seem to be compressed by the hyperplastic connective tissue of the walls of the artery at large; hence the circulation in them has probably been impeded.

The small arteries show the increase in the adventitia to a lesser degree than those just described, but the media shows a very scant supply of muscle cells, and is largely replaced by the hyperplastic tissue; in addition there is a diffuse hyaline degeneration of the coat in many of the vessels. This change also extends into the subendothelial coat and involves the fibroblasts which are found quite abundantly in the smaller trunks. Swelling, and in places desquamation of the endothelium, is present, particularly over those parts where the degeneration of the subendothelial coat is most extensive.

Many of these smaller trunks are filled by clot, a considerable portion of which is of the white variety, but as a rule their lumen is distended and filled with blood.

The capillaries are mostly congested with blood. The lymph space about them is generally dilated, and in a few cases rupture of the capillary wall has permitted the extravasation of a few cells. In some cases the endothelium of the capillaries seems to be in a state of karyokinesis. A good many capillaries are plugged by clot.

ACUTE ARTERITIS OCCURRING IN THE VESSELS OF THE CENTRAL NERVOUS SYSTEM IN RABIES.

J. B., aged thirty-two years. Machinist. Admitted to Hood Wright Hospital April 16, 1902, 9.20 p.m.

Family history.—Unimportant.

Past history.—Two months ago (February 15 or 16) patient was bitten by a small fox terrier, which gave no sign of being rabid. The bite took place in the fleshy part of the right thumb. The patient sucked the wounds as soon as the dog let go but paid no further attention to them; they healed within a week.

Present history.—April 15 patient noticed an itching or pinching sensation in the right hand about the old wounds and felt chilly. April 16 he complained of some pain in the hand and shoulder of the same side and began to have some difficulty in swallowing liquids.

Examination.—Perfectly conscious, rational, and shows no excitement. No paralysis. Sensation of numbness in right hand and fingers. Heart, lungs, liver, spleen and kidneys, negative. T, 99.6; P., 72; R., 24. Patient drinks with great difficulty and only after considerable exertion.

The patient finally developed the typical symptoms of rabies, failed to respond to the most energetic treatment and died April 18, two days after admission. In the latter stages of the disease he showed marked hypersecretion of saliva, and the temperature rose to 104.2 degrees. The pulse became very rapid and feeble and blood count showed a leucocytosis of 26,000.

Post-mortem examination showed in brief very few gross changes aside from a slight arteriosclerosis of the general vessels, acute lymphangitis and pulmonary edema.

Microscopic examination demonstrated very few changes in brain and cord except chromatic alterations in the ganglion cells, particularly those of the medulla, and the alterations of acute lymphadenitis. The blood vessels of the central nervous system show the marked changes of acute arteritis to be described in detail.

Experimental inoculation of rabbits with emulsions of the medulla and cord caused the development of rabies in these animals. The case was thus demonstrated to be a typical one of rabies.

The changes noted in the arteries are most marked in the medulla, though they are also present, but in considerably less degree, in the vessels of other portions of the brain and cord. A good many of the arteries show absolutely no change whatsoever. The medium sized vessels are but slightly involved, the chief changes being found in and about the small arteries and arterioles. The perivascular lymph space is markedly dilated in almost every instance. The connective tissue cells of the supporting tissue, of the scant adventitia and of the intima show, almost without exception, frequent evidences of active cell division, and the endotheloid cells seen in the perivascular lymph spaces also exhibit frequent karyokinetic figures. There is a very marked exudation of leucocytes, chiefly small mononuclears, into the walls of the vessels, where they separate the fibers and muscle tissue and infiltrate into the lymph spaces, where they mingle with desquamated fibroblasts and proliferating endothelial cells. The endothelium of the intima also shows evidences of active cell division. Polynuclear leucocytes are found about the periphery of the channels, where they are united to the eroded endothelial wall by shreds of fibrin. Notwithstanding the fact that the walls of many of the vessels are seriously damaged, diapedesis of the erythrocytes is not shown. There is a slight mesh work of fibrin present throughout the vessel walls.

Changes of like character, but of less extent, are also found about the capillaries but, as is the case with the arteries, they are not universally involved.

THE TREATMENT OF CEREBRAL PALSIES AND ATHETOSIS BY NERVE ANASTOMOSIS AND TRANSPLANTATION.[1]

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AND

Charles H. Frazier, M.D.,
PROFESSOR OF CLINICAL SURGERY AND SURGEON TO THE UNIVERSITY HOSPITAL IN THE UNIVERSITY OF PENNSYLVANIA.

REMARKS BY DR. SPILLER.

The views presented in this paper are put forward with much hesitation, because the means by which voluntary power is restored in hemiplegia are unknown, and the conditions have resisted most attempts at improvement. The views here expressed are original with the authors, and if experience should prove that they are fallacious, they may nevertheless afford some suggestions for further investigations. We should have preferred to wait until these investigations had been carried to a successful conclusion before making any reference to them, but it may be better to invite discussion on the subject.

Is it possible to benefit in any way by surgical means the patient who is afflicted with an incomplete hemiplegia? This is a question I have repeatedly proposed to myself. Where the hemiplegia is complete or nearly complete we can not hope to accomplish anything by any surgical procedure. There are, however, many cases in which there is a partial return of power in the paralyzed limbs, and experience shows that usually the restoration of motion is greater in the flexors in the upper limb and greater in the extensors in the lower limb, except those of the toes. I have—to give one example—under my observation at present a man, aged twenty-seven years, who has had left hemiparesis so long as he can remember. He has excellent power in the flexors of the hand, but can extend the hand and fingers only to the plane of the forearm, and he has no contractures. In the lower limb he can not extend the toes or dorsally flex the foot, although he can extend the foot with much power, and in walking raises the left knee a little more than the right, because the left lower limb is too long. An operation on the upper limb would not be permissible, because the flexors of the fingers are more useful than the extensors, and he can extend the fingers to a level with the back of the hand. In other cases, however, the difference in the degree of power of the flexors and extensors of the fingers and hand is greater, and the restoration of power may occur almost alone in the flexors. The latter may be contractured so that the hand is flexed on the forearm and the fingers flexed into the palm. The restoration of the power of the flexors of the fingers and hand may be of little or no value, unless there is at least a partial return of power in the antagonistic muscles, although this return need not be so great in the extensors as in the flexors. I have thought that in a case where the flexors alone regain power we might anastomose the central ends of some of the least important of the flexor nerves with the peripheral ends of extensor nerves, and in this way restore more nearly the normal relation between the flexor and extensor muscles. In the case I have referred to above, in which the flexors of the toes were much stronger than the extensors, an anastomosis of the central ends of one or more flexor branches with the distal ends of extensor nerves might be of decided advantage. Even though the hemiparesis has existed many years, regeneration of the united nerve ends would occur, because these nerves still have power and their connection with their spinal cells of origin has never been impaired. The muscles of course must not be entirely wasted.

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[1] Read by invitation before the New York Neurological Society, March 7, 1905.
From the Laboratories of Neuropathology and Surgical Pathology in the University of Pennsylvania.

The question now arises, would impulses pass from the brain over the central motor tracts to the anastomosed fibers in such a way that useful return of function might be expected? I think the probability is that such a restoration in part at least would occur. It is true that movements and not muscles are represented in the brain cortex, but it has been demonstrated that when the lesion is in the peripheral nerves and anastomosis of nerves has been performed, a new form of associated movements may be learned by the brain.

We need not enter into a discussion of the manner in which return of function occurs after hemiplegia. Whether there is a close association in the brain of the fibers for the agonistic and antagonistic muscles, to use the terms employed by certain German writers, as Mann believes, need not concern us now. The partial return of power after a hemiplegia, according to the view of Rothmann, may be the result of independent function newly acquired by subcortical centers, especially by the optic thalamus, and is such as exists normally in lower animals, or is developed in the monkey a few days after destruction of the cortical centers for the limbs.

It is certain that for perfect voluntary motion we must have return of power in both agonistic and antagonistic muscles. We can not flex a finger properly unless the extensor of that finger is in the requisite degree of tonicity. If we flex the fingers of one hand firmly into the palm and place the other hand over the extensors of the wrist, we find that the extensors of the carpus become tense. It is in large measure because of this loss of extensor power, and thereby of fixation of the hand, that flexion of the fingers is weakened when we have wrist drop from a lesion of the posterior interosseous nerve.

In order to study the conditions of paralysis existing in hemiplegia I have examined twenty-six cases in which the paralysis dated from early childhood. Dr. Martin W. Barr kindly had collected for me all the cases of infantile hemiplegia at present in the Pennsylvania Training School for Feeble-Minded Children. I wish to thank Dr. Jennings and Dr. Weisenburg for their assistance in the study of these cases.

The return of power is, I think, greater on the average in cases in which the hemiplegia develops in childhood than in those in which it develops in adult life. These twenty-six cases were not selected from a large number of hemiplegics, and yet in eleven of these the voluntary power in the flexors of the hand and fingers was much greater than in the extensors; and in four of these the voluntary power of the flexors of the toes and extensors of the foot was much greater than in the extensors of the toes and flexors of the foot. The cases in which the hand had regained partial power were more numerous than those in which the foot had regained partial power. In eleven cases out of twenty-six, therefore, the flexors of the hand were so much stronger than the extensors that a nerve anastomosis might properly be attempted, but only four cases offered much hope for improvement from an operation on the foot. All cases in which the restoration of power in the extensors of the hand and fingers was fair, even though less than in the flexors, were rejected as being undesirable for operation. As one of the most suitable cases the following may be briefly described:

L. A., female, aged thirty-one years, high grade imbecile, has voluntary movements at all parts of the right upper limb, but the movements are impaired. The fingers of the right hand are slightly flexed into the palm. She has considerable power in the flexors of the right hand, but no power in the extensors. In making a bed she is able to hold the bed clothing with her right hand, but the use of the hand is much diminished by the permanent flexion. The paralysis in the muscles of the right foot is almost complete.

There is probably therefore a field for surgical intervention in certain cases of cerebral hemiplegia, but the cases must be carefuly selected, and each thoroughly studied. Whether any benefit by nerve anastomosis will result to the hemiplegic individual or not, experience alone will decide. The subject, however, is one deserving attention. Dr. Frazier has in one case operated upon the hand of a partially hemiplegic woman, but the return of power following the hemiplegia has not been sufficient until the present time to make the intervention entirely satisfactory. He will describe his method of anastomosing the nerves. He has also made investigation upon dogs concerning the results of anastomosing flexor nerves with extensor nerves.

Tendon transplantation for the increase of function in paralysis of all varieties has proved of benefit provided the paralysis is not progressive,—a subject upon which Oppenheim has written within the last few weeks—but where nerve anastomosis or transplantation is successful we have a means of restoring the function of muscles previously paralyzed, and therefore this method is better than tendon transplantation which makes no demand on paralyzed muscles.

I am well aware that improvement may occur in hemiplegia years after the paralysis has first developed.

Athetosis is one of the most distressing forms of involuntary movement, and the failure to control it in any degree is a reproach to medicine. I have for several years been trying to devise some method of treatment for this condition. Much study has been given to the causation of these movements, but our knowledge regarding the condition is very insignificant. There must in cases in which athetosis exists be an irritation of the motor system somewhere, and the investigations of recent years (Bonhoeffer, Mann) seem to point to implication of the superior cerebellar peduncle. We can not hope to remove the irritation in the brain. We can not hope to cut the central motor fibers. Such a procedure would be unjustifiable. Can we accomplish anything by operation upon the peripheral nerves?

Where athetosis exists the muscles are in a state of greatly increased tonicity and often are abnormally well developed and abnormally strong. Theoretically the proper procedure might be to cut posterior roots of the affected limbs, the number to be cut depending on the condition in each case; but this is always a serious operation, and the results have at times been unexpectedly grave.

It is possible that if we were to divide one or more of the motor nerves of the affected limb and immediately suture the divided portions we might lessen the involuntary activity and weaken the muscles only slightly; or if experience taught us to make the paresis greater by making the conditions for full restoration of power less favorable, we might be justified in leaving some of the nerves divided, or even in making reunion of certain ot the nerves improbable. It is possible that in this way we might diminish the nervous impulses sent out continually and involuntarily from the brain, or at least check their manifestation. In athetosis the flexors of the hand and fingers are often unusually strong, and where this is the case the union of the peripheral end of a flexor nerve with the central end of an extensor nerve and *vice versa*, might restore the proper relations of voluntary power and lessen or abolish the involuntary movements. We have at present a case of marked flexor spasm with athetosis on which Dr. Frazier will operate.[2]

Nerve transplantation if the lesion is in the spinal cord is a hopeful field for surgical intervention when the conditions are favorable. I[3] have already discussed this subject in association with Dr. Frazier, in a paper read before the Section on Nervous and Mental Diseases of the American Medical Association in June, 1904, and may say here that paralysis confined to one or a few muscles is not an uncommon occurrence in anterior poliomyelitis, and that the cases in which nerve anastomosis may be done are therefore fairly numerous.

REMARKS BY DR. FRAZIER.

The adaptation of the operations known as nerve anastomoses or implantations, to the treatment of cerebral palsies, as proposed by Spiller, opens up a field in neurological surgery which seems to me to be full of promise. There are innumerable details in the method of executing the operation, which can be determined only by thorough and repeated experimental investigations and clinical observations. A series of experiments is now being conducted by us, but few have as yet reached the stage which warrants their publication. Our first clinical experience dealt with a woman 65 years of age, who five years ago had an apoplectic attack. When the operation was performed, July 28, 1904, she was hemiplegic; there was marked atrophy of the muscles of the hand, forearm and arm, but the paralysis was so much more marked in the extensor than in the flexor group of muscles that she could partially, and this only with great difficulty, extend the proximal phalanges. Her hand was a greater part of the time held in partial flexion. Under ether anesthesia an incision was made along the inner border of the coraco-brachialis, exposing the brachial vessels and the main nerve trunks. The median and musculo-spiral nerves were isolated sufficiently for our purpose and a flap, composed of one-half of the median nerve, with its base centralwards, was implanted into the musculo-spiral. Union was effected by making a longitudinal incision in the sheath of the musculo-spiral into which the free end of the flap from the median nerve was introduced and sutured in such a way that the nerve fibers of the median were imbeded into those of the musculo-spiral. The results of the operation up to the present time have not been very positive although it is still possible that as time goes on greater improvement will be noted. There is probably distinct improvement in the extension of one of the fingers. It is too early yet to predict the result.

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[2] Since this paper was read nerve transplantation has been done and the patient's condition is distinctly improved.

[3] Journal of the American Medical Association, Jan. 21, 1905.

The experimental work is being carried out on dogs and has consisted essentially in anastomosing the nerves presiding over one group of muscles with those of another. Thus for example, in several instances the peripheral end of the divided sciatic nerve has been joined to the central end of the divided anterior crural, and the peripheral end of the anterior crural with the central end of the sciatic. In these cases the operation was followed by transitory paralysis, but during the third week the animals began to regain power in the affected limb. At first in their attempt to walk the affected limb was extended with a jerky movement, instead of being flexed as it should, and after three or four attempts at using the affected limb the animals apparently became discouraged and ran around on three legs. About a week later the animals had apparently learned to control the limb, at least when walking, although in attempting to run they again became confused and the limb would be extended, when it should have been flexed. Before two months had elapsed, however, these incoordinate movements had disappeared.

These experiments are cited simply as illustrating the lines along which the experiments are being conducted. Many modifications of this plan will of course have to be worked out in detail before the experiments will be reported in full. As compared with tendon transplantation, the only other surgical procedure which can be considered as an alternative, nerve anastomosis, theoretically at least, is the more rational measure. If one is able to restore function to the paralyzed muscle, the functional results should be better than if an attempt is made to compensate for the loss of function of one group by putting an additional tax upon another.

Applied to the treatment of those palsied conditions with uneven distribution of power, in those muscles, which oppose one another, I cannot but feel that in the transference of a proportion of nerve force, in an amount still to be determined, from one group of muscles to another, from the stronger to the weaker, we have at our disposal a means of control which may be productive of gratifying results.

THE LOCATION WITHIN THE SPINAL CORD OF THE FIBERS FOR TEMPERATURE AND PAIN SENSATIONS.[1]

By William G. Spiller, M.D.,
of Philadelphia,

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Many believe that the tracts of Gowers may contain the fibers for the sensations of pain and temperature, but evidence supporting this opinion has been sadly deficient. There has been little to show that the fibers for these sensations are confined to Gowers' tracts and are not present elsewhere in the antero-lateral columns. J. Piltz[2] is the most recent author to support the view referred to above.

A case has been in my wards at the Philadelphia General Hospital within the last few months which seems to me to afford the best evidence that has as yet been offered for the location of the fibers for temperature and pain within the tracts of Gowers.

A man, twenty-three years of age, began to have pain in the lumbar region and abdomen four months before he came under my observation, Sept. 1, 1904. Numbness was soon felt in the feet, and gradually involved the lower limbs below the knees. When standing he had extreme genu valgum, which had existed about five years. He had scars on the abdomen which he said were caused by painless burns in childhood. Resistance to passive movement was slightly diminished in the lower limbs. Sensation for touch was normal or nearly normal in the lower limbs, but sensation for pain was abolished or almost abolished in these parts. Sensation for temperature was almost lost in the lower limbs below the knees, and much impaired in the thighs and lower part of the trunk. Walking was difficult because of the deformity of the knees.

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[1] Part of a paper read by invitation before the Section on Neurology and Psychiatry of the Medical and Chirurgical Faculty of Maryland, March 8, 1905. The paper will be published in full in the University of Pennsylvania Medical Bulletin.

[2] Pilz. Neurologisches Centralblatt, March 16, 1905.

He had a slight kyphosis at about the tenth thoracic vertebra. The upper limbs and face were not affected.

After a fall backwards down a flight of steps he became completely paralyzed in the lower limbs. Sensation for touch was then lost in the right lower limb for a time, but was preserved in the left lower limb. Sensations for temperature and pain were lost in the right leg and foot and in the left foot, but were preserved elsewhere, although it is not stated by Dr. McConnell, who made this note, that they were normal elsewhere. The patellar reflexes were much exaggerated and Babinski's sign was present.

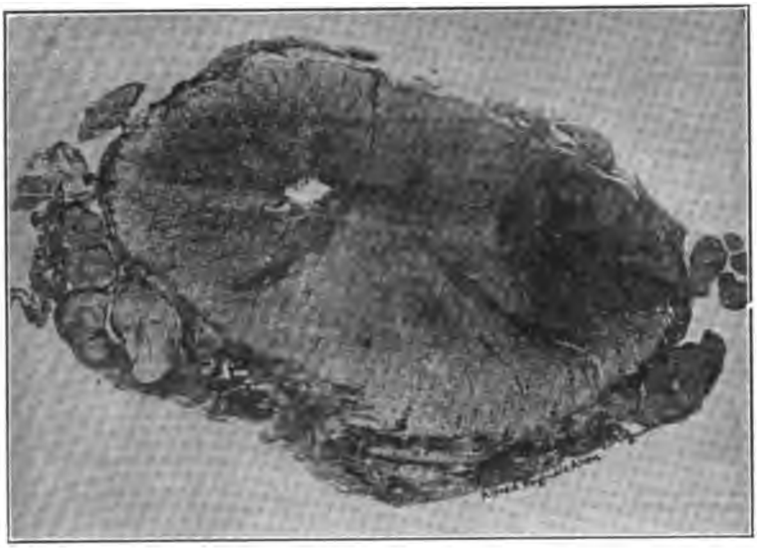

Fig. 1. Small tubercle in the right lateral column extending forward into Gowers' tract.

Still later sensation for touch was found preserved everywhere in the lower limbs. Sensation for pain was irregularly present in the lower limbs, the response to pain stimulation being very uncertain, and sensation for temperature also was much impaired in the lower limbs.

Tuberculous meningitis and caries of the vertebræ were found, and a small tubercle was present in the right lateral column at the extreme lower end of the thoracic cord, involving the area of Gowers' tract. About one-half to one inch higher another small tubercle was found involving the left tract of Gowers. Although there was some myelitis, it was very evident from the symptoms that it had chiefly developed after the fall down the flight of steps, as at the time the patient first came under my observation weakness in the lower limbs was slight. He lived four months after the accident.

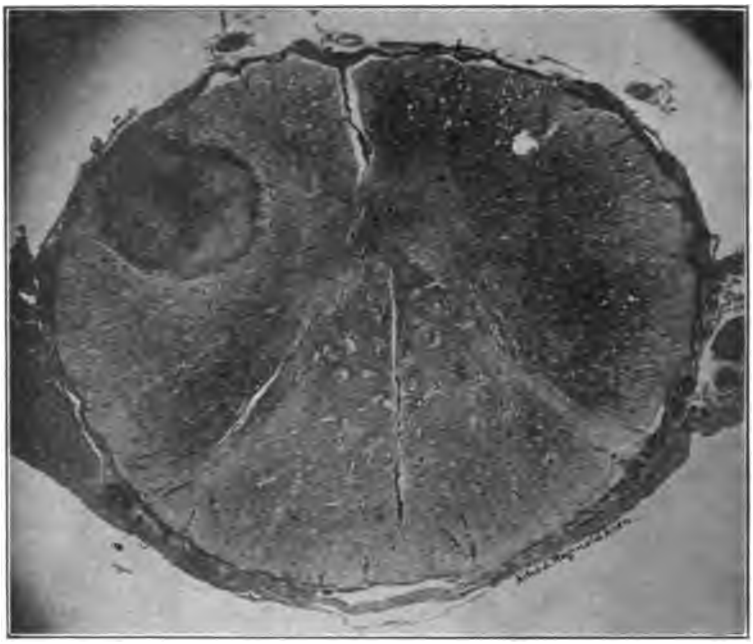

Fig. 2. Small tubercle in the left antero-lateral column implicating Gowers' tract.

THE CURABILITY OF EPILEPSY.

By Jas. W. Wherry, M.D.,
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The subject of the curability of epilepsy is not one to be lightly dismissed; nor, on the other hand, is it one for which any extravagant claims will be tolerated. To profess to cure any large per cent. of cases, taken at random and without judicious selection, is to invite suspicion and distrust; but, at the same time, to waive all cases aside as incurable, without discrimination or distinction, is an indication of professional apathy and indifference, and of a disposition to excuse any possibility of failure in advance by a widespread and all-embracing declaration of the futility of any attempts in this direction. No physician ever cured a case of illness, no matter what its nature, that he did not *expect* to cure. If the patient recovers at all under such circumstances he recovers in spite of the doctor. Faith and confidence in the method of treatment adopted in any disease is as necessary to the physician as it is to the patient. Faith and confidence born of a knowledge of drugs and a knowledge of the disease, and a knowledge of the effect of the one upon the other, is the physician's stronghold, and herein lies his power; and the patient's faith and confidence lies in the unfaltering belief that the physician possesses, not only this knowledge in the abstract, but that he has faith and confidence in it as well.

Sweeping assertions regarding the curability of epilepsy cannot be made, for but few men, indeed, have brought to the task all the necessary characteristics, or have employed these under the most favorable circumstances. The very nature of the disease has precluded its admission to general hospitals and to the individual treatment and consideration it so sorely needs, and few of the ills to which mankind is heir has received so much theoretical attention and so little real professional care. From time immemorial it has been the most prolific field for quacks and vendors of patent nostrums, and why? Simply because it had been so nearly abandoned by the profession. Let the profession once lose faith in the cure or amelioration of any disease, and there will quackery flourish like a green bay tree. Pronounce cancer incurable, and the patient goes elsewhere for relief. Pronounce epilepsy incurable, and the advertising pages of literature are flooded with promises and assurances of cure. Pronounce tuberculosis incurable, and the same result will follow. Pronounce any disease incurable and what follows? Does the patient sit down and fold his hands in despair? By no means. He goes where relief is promised. To him, one man's promise is as good as another man's pronouncement, and he is frequently justified in his faith, for experience has shown that so-called incurable diseases have frequently been cured.

If a single cancer has been cured, cancer can no longer be called incurable. If a single epileptic has recovered, epilepsy can no longer be classified as an incurable disease. If small tubercular foci ever spontaneously heal: or if more pronounced invasions sometimes recover, then tuberculosis can never be termed incurable because the long-neglected and more advanced cases invariably die.

Cases of epilepsy have been cured. Not many it is true, compared with the large number afflicted, but a very respectable per cent. when everything is taken into consideration. Enough, at least, to forever bar the word "incurable" in connection with epilepsy. That more have not been cured is due in a great measure to the fact that for many years the convulsion has been mistaken for the disease, and treatment has, consequently, been misapplied. We are beginning to see our way clearer now and by reason of these new truths the prospects for recovery in epilepsy are becoming brighter every day. Failure to cure epilepsy does not necessarily mean that epilepsy is incurable, for failure in the past has been based upon, and due to, an insufficient knowledge of the disease, and the almost utter impossibility of intelligently putting into effect the few epileptic truths we did possess.

The ideal treatment of epilepsy, and there is an ideal treatment, is practically impossible under ordinary circumstances. In the first place, epilepsy is specifically and distinctively an individual disease and must receive individual care and consideration. The tendency in the modern study of medicine is to systematize and classify, and in the attempt to reduce diseases into specific allotments, and to then formulate a general treatment which is to apply to all of each class, the individual is frequently forgotten and always overlooked. To such an extent has this been carried that, whenever a patient in any particular class of diseases fails to respond in the usual way to the prescribed drug, he is said to possess an "idiosyncrasy," and the doctor regards it as little less than an affront, and, indeed, is often inclined to remonstrate with the patient for casting such an unnecessary reflection upon the established facts of medicine. The truth is that individuals existed long before the science of medicine, and as God in His wisdom saw fit to make no two of them alike it is not to be wondered at that they do not respond identically to the same drug. On the contrary, it is a constant source of wonder that so many respond so nearly alike as they do in some diseases.

That all epileptics do not respond to the same drug in the same manner should not occasion surprise. Epilepsy is not a disease founded upon general principles which apply in all cases alike. Its fundamental element is its individuality, and this one fact has done more to baffle investigation than anything else. That the facts established in one case so conclusively would not apply to another, was disturbing and tended to reflect doubt and suspicion upon the observations made. When the results of study and investigation in a given series of cases were more or less contradictory and confusing, the observer became discouraged and disheartened and in many instances was prepared to assume a position of general distrust upon slight provocation. We have found, however, that the study of epilepsy must be individual in its nature; that each patient is a case in himself, and that no two can be successfully treated from the same formula in every particular.

In epilepsy there must be individual treatment if recovery is to follow. Not that all cases of epilepsy treated individually will recover. Epilepsy, like other diseases, has its recoverable cases and its non-recoverable cases, but recovery, even in recoverable cases, is not likely to follow routine treatment with a single formula for all. There is no known specific treatment for epilepsy and no one method that can be recommended in all cases, and success in the treatment of epilepsy comes from the study, by the physician, of each separate case, and the intelligent application of the method best adapted to each individual. The treatment of epilepsy cannot be learned from text-books—it must come from experience born of a desire to know the truth, and an unflagging study of the morbid physiological conditions present.

To make an individual study of each case and to carry out an individual treatment in each instance is impossible with a large number of patients. The number must be limited if individual care is to be given by the physician. For this reason it might be thought that the treatment of epileptics at home would be an ideal arrangement, but there are features pertaining to the treatment of epilepsy which must not be overlooked and which militate against treatment at home. Individual medical treatment is but one element in the care of an epileptic. Another is, diet. So much has been written regarding the diet of epileptics that there seems to be nothing left to say, but the same error has been committed in prescribing the diet of epileptics as in administering drugs. Epileptics are always considered *en masse*, as though there was but one thing in all physiology or pathology capable of causing convulsions, and if this One Thing could only be subdued by medicine or abolished by diet all would be well. Convulsions, however much they resemble each other, are not always the offspring of the same One Thing. Many conditions produce convulsions, and even labeling convulsions as "epileptic" does not signify that they are all the product of One Thing. Consequently, because the elimination of a certain article of diet has been productive of good results in a certain case, we are not to conclude that this specific elimination applies to all cases, or, for that matter, to any other case. There must be the same individual study of diet as there is of drugs.

This fact alone makes it all but impossible to properly care for an epileptic at home, and makes it equally difficult where large numbers of epileptics are congregated and where many eat in a general dining room. The diet of the epileptic cannot be regulated at home, even though the patient expresses his willingness to coöperate in the measures taken, any more than a man will voluntarily starve himself, in the midst of plenty, simply to reduce his weight. He will, or may, do so for a time, but the appetite of an epileptic is proverbial and beyond the patient's control. Even in institutions for the treatment of epilepsy this regulation of the diet is one of the most difficult matters encountered, and even here, patients for whom a strict regulation of diet is necessary should not eat at a common table (where the differences in the diet prescribed is so plainly in evidence and where, in many instances, the patient cannot see the necessity for it) but from a tray in the privacy of his own room. Unless such precautions can be taken, and unless the diet of the patient is absolutely and unquestionably in the hands of the physician, little can be expected from the treatment of epileptics at home, and no case of epilepsy, no matter how excellent the medical care given, should be pronounced incurable until such dietetic measures have been employed.

But individual medical treatment and individual supervision of diet are not enough. There are various forms of dissipation and of personal habits which, while not causing epilepsy in themselves, can successfully prevent a recovery so long as they exist, and it frequently becomes as necessary to forcibly separate the epileptic from his environment as the alcoholic or the morphine fiend from his source of intoxication. It is needless to expect recovery on the part of an epileptic in the absence of a clean life, for his mental, moral, and physical surroundings have more real, actual influence upon his well-being than is found in any other class of diseases except insanity. One of the chief characteristics of the epileptic is his mental, moral and physical instability, and this feature must receive serious consideration if a recovery is hoped for. The convulsion is not all that there is of epilepsy. To suppress the convulsion by the mere chemical force of drugs is accomplishing no real good if the patient is to become an imbecile or a dement as a result. To suppress the convulsion is of no avail if the patient is to become a physical wreck by so doing. The treatment of epilepsy is not to be conducted on a single, narrow line, with an eye singled to the convulsion alone. It must be broader, and must embrace the whole individual, mentally, morally, and physically, and until it is so considered medical treatment and supervision of diet alone will not prove effective in producing a cure.

I would suggest, then, as the treatment of epilepsy: A study of each case individually; special adaptation of drugs to individual conditions; personal supervision and individualization of diet; absolute change of environment. These are the difficulties to be met, and when these have all been encountered and successfully overcome then will be the time to voice an opinion as to the recoverability of epilepsy. To accomplish all this requires months or years of time and unlimited patience, but until we can bring to bear upon the situation not only time and patience, but scientific intelligence and a faith and confidence in medical resources as well, we cannot hope for recoveries; and it is unscientific to pronounce a disease incurable simply because, by reason of our limited knowledge of the real conditions present, we have neglected to use all the means at our command and have, consequently, met with comparatively small success.

That epilepsy is by no means incurable is amply demonstrated by Nothnagel, Turner, Spratling, Alt, and others, and many of the results given by these investigators were obtained under the most unfavorable circumstances. Ideal hospitals for the treatment of epilepsy are far from numerous, and experimentation and investigation has hitherto of a necessity been carried on in most instances under conditions which in their very nature did not favor a large percentage of recoveries. Epileptics treated at home, or in county houses, or in large colonies or homes, where individual treatment is impossible, cannot be expected to show so many recoveries as if they had been cared for in hospitals especially constructed for the treatment of this disease, and the fact that so many have recovered in the absence of special provision only indicates, in a small way, the possibilities which may be legitimately expected in the future.

Spratling says, in The Tenth Annual Report of the Managers and Officers of the Craig Colony for Epileptics: "The great majority of epileptics admitted to the Colony are palpably incurable on admission. Less than 1½ per cent, of them have had the disease less than a year, while many have had it for 30 or 40 years. Fully 50 per cent. who enter show mental impairment in some form or degree, being demented, feebleminded, imbecile, idiotic or insane. * * * Taking the 1,286 cases admitted to October 1, 1902, and deducting one-half as wholly incurable, and we have 643 more or less chronic cases left. From this number we can report 16 as cured, all of them having gone from two to five years or more without an attack; and 15 others who bid fair to make a complete recovery, these having gone a year and a half or over without an attack. Add these and we have 31, practically 5 per cent. of the possibly curable cases. It seems fair to say that even with the chronic cases we receive, we may expect recovery in 5 per cent.

"If one-half of our admissions could be within the first year or so of the disease, the number of recoveries could be doubled. If all could be taken within that time, a still larger percentage of cases could be expected.

"This is one reason why the recoveries in insanity exceed those of epilepsy. The insane person is at once placed under proper treatment. The form of his malady demands this. The epileptic is not often troublesome at first and is temporized with at home until his malady is beyond eradication, then he is sent to some institution. Too often he is dosed with patent medicines at home that in the end do him enormous harm. Scores of such cases come to my notice annually."

If 5 per cent. of the curable cases recover, or, even 3 per cent, or 1 per cent., it can scarcely be said that epilepsy is an incurable disease. In its very nature epilepsy tends to chronicity. It is due to a faulty metabolism which, when established, perpetuates itself, in the absence of proper treatment. That old, chronic, well-established cases do not recover is no cause for surprise. Epilepsy does not stand alone in this respect, nor does it differ from many other diseases in yielding more readily to treatment in its incipiency.

That there are so few recoveries in epilepsy is due to the following reasons, namely:

1. An insufficient knowledge of the real conditions present.
2. Treatment of the convulsion, instead of the epileptic condition as understood.
3. Treating epileptics *en masse*, instead of individually.
4. Treatment postponed until disease becomes well-established.
5. Limited provision for special care and treatment.

In spite of all this, however, it is gratifying, as well as inspiring, to know that epilepsy, instead of being an incurable disease, has, under the most unfavorable circumstances, shown a schedule of recoveries in excess of some other diseases and which in the future will be made much larger. Recoveries in epilepsy reported by recent writers are: Nothnagel, 5 per cent.; Laehr, 6 per cent.; Ackerman, 7.6 per cent.; Wildermuth, 8.5 per cent.; Dana, 5 per cent. to 10 per cent.; Turner, 10.2 per cent.; Habermaas, 10.3 per cent.; Alt, 12.5 per cent.

I do not wish to be regarded as over-sanguine, nor do I make the statement without due consideration, based upon some experience; but I have every reason to believe that individualization of treatment in epilepsy, with change of environment, will result in doubling the percentage of recoveries already obtained, and that this final result may again be doubled by a judicious selection of cases. The treatment of epilepsy is really in its infancy. We have been seeking a specific for convulsions, and the majority of the drugs used in the past have borne no more relation to the epileptic condition than the coal tar products bear to typhoid fever. We have since learned that the rise in temperature is not the most dangerous thing in typhoid fever; that it is but a symptom of the disease. We have also learned, or are learning, that the convulsion is but a symptom of the epileptic condition, and that the coal tar antipyretics are no more objectionable in typhoid fever than antispasmodics in epilepsy. More than this, we are learning that, under proper treatment epilepsy is curable, and it will become more and more so as hospitals for the special treatment of epilepsy multiply.

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

December 6, 1904.

The President, Dr. Pearce Bailey, in the Chair.

A Case of Exophthalmic Goiter, Associated with Scleroderma and Alopecia Areata.—Presented by Dr. Frederick Peterson. The patient was a single woman, 25 years old, a music teacher by occupation. She enjoyed excellent health until the age of 20, when she developed goiter. This was the first symptom noted, and subsequently the exophthalmus and tachycardia appeared. When she first came under Dr. Peterson's observation, early in November of the present year, the proptosis was marked. The pulse ranged from 90 to 120. About eighteen months ago a patch of scleroderma developed over the right hypochondriac region; this was 6x12 centimeters in dimensions. Soon afterwards a second patch appeared on the right breast, which now involved a considerable portion of the skin of that organ. Subsequently a third patch appeared under the left axillary space, a fourth in the left supraclavicular region, and a fifth in the left lower abdominal region. There were no sclerodermatous patches on the face or extremities.

About three years ago she developed a bald spot on the top of her head, about 5 centimeters in diameter. She now had three such patches of alopecia areata. There was no specific history and no hereditary taint.

Möbius states that von Leube, about 1875, was the first to record his observations of scleroderma of the face and hands in a patient with Graves' disease. Von Leube, in his book on "Medical Diagnosis," New York, 1904, says that in Graves' disease sclerema of the skin has often been observed by himself and others. Kahler, in 1888, reported a case of scleroderma with exophthalmic goiter. Jeanselme, in 1894, reported cases of scleroderma in Graves' disease. G. Singer, in 1894, stated that scleroderma frequently occurred in connection with diseases of the thyroid gland. He found that organ usually affected in ordinary scleroderma. Beer, in 1894, reported four cases of scleroderma, in all of which there was tachycardia, and the volume of the thyroid was diminished. Ditisheim, writing on Graves' disease in 1805, said that 45 per cent. of the cases observed by him in Zurich had scleroderma in addition to Graves' disease. Grunfeld, in 1896, reports a case of Graves' disease with scleroderma. Ord and Mackenzie, writing on Graves' disease in 1897, said that the association of scleroderma and Graves' disease has been recorded by several observers. Also, that alopecia has been recorded. Osler, in 1898, reports a case of a man with Graves' disease and scleroderma. Raymond, in 1898, in a lecture on scleroderma, presented two patients with scleroderma and Graves' disease. Dupré and Guillain, in 1900, reported the case of a man with Graves' disease, scleroderma and sclerodactyly. Kriger, in 1903, reports a case of a woman with sclerodactyly and Graves' disease.

As regards the relation of alopecia areata to Graves' disease, there is not so much reference to it in literature. It was mentioned in one of the cases already cited, and Dore, in 1900, writing on cutaneous affections occurring in the course of Graves' disease, refers to the frequent loss of hair, and says: "Alopecia areata is occasionally seen; Mr. Malcolm Morris has had two cases under his care." Stelwagon, in his book on "Diseases of the Skin," 1904, makes a casual reference to the association of Graves' disease with alopecia areata, in considering the pathology of alopecia. Luithlen, in his "Handbuch der Hautkrankheiten," 1904, refers to alopecia as an occasional complication with scleroderma.

We have, then, the fact that it is not infrequent to meet scleroderma in association with Graves' disease; that sometimes scleroderma is associated with alopecia and that alopecia is sometimes met with in Graves' disease. In this patient we have a combination of Graves' disease with scleroderma and also with alopecia areata.

Brain-Tumor (?) Two Cases of Doubtful Etiology.—Presented by Dr. William M. Leszynsky.

CASE I.—Sarah Z. U. S., single, 28 years old; a stenographer and typewriter by occupation. When she was first seen, in December, 1903, she complained that for six months previously she had suffered from frequent paroxysmal attacks of severe frontal and occipital cephalalgia, with vertigo, nausea and vomiting. The frontal headache was continuous and often prevented sleep. Her vision began to fail, especially in the right eye, and two months later that eye became blind. Soon afterwards the sight of the left eye was also lost. There was no history of injury to the head, alcoholism or syphilis. In childhood she had measles and diphtheria, and in her second year scarlet fever and right suppurative otitis. Menstruation began at the age of 15, and was regular during the first year. It then appeared at irregular intervals of from four to six months, and during the past year there had been amenorrhea. There was chronic constipation. The family history was unimportant. An examination of the blood showed 70 per cent. of hemoglobin; no leucocytosis. The pupils were dilated and rigid. The motility of the eyeballs was normal. There was no perception of light. Bilateral papillitis. 5 D. No retinal hemorrhages. No evidence of a kidney lesion or renal inadequacy. There was occasional right facial paresis of the lower branches of the nerve. After remaining under observation in the hospital for one week, she was discharged. Subsequently, she was trephined by Dr. Andrew McCosh at the Presbyterian Hospital. No improvement followed the operation, which failed to reveal the presence of a neoplasm. An X-ray picture of the skull was negative. There was no improvement under increasing doses of potassium iodide. The blindness persists, the disks having become atrophic.

CASE II.—Male, 28 years old, a native of Russia and a photographer by occupation, was admitted to the hospital in October, 1903. For several months he had suffered from frequent attacks of severe generalized headache, preceded or accompanied by vomiting. Soon afterwards he became blind, and complained of weakness and vertigo, with the sensation of falling to the right. His father died of diabetes; his mother was alive and well. During childhood the patient had suffered from measles and scarlatina. He was addicted to the excessive use of whiskey, beer and wine. He admitted having had gonorrhea, but denied syphilis. There was no history of injury to the head. An examination showed paralysis of the right external rectus. Both pupils were dilated and rigid, and there was no perception of light in either eye. Bilateral choked disk of 6 D., with numerous retinal hemorrhages. There was left hemiparesis, and occasional flexor rigidity in the left upper extremity. Pronounced astereognosis (fluctuating), and slight ataxia. No disturbance of tactile, pain or temperature sensibility. Both lower extremities were extended and rigid from time to time, with spurious ankle clonus and trepidation. Left knee-jerk exaggerated; both plantar reflexes exaggerated. No Babinski. Other reflexes normal. Urine, blood and X-ray examination negative. The patient was put on increasing doses of potassium iodide, and two months later all the symptoms disappeared, but the blindness persisted. The retinal hemorrhages had become absorbed and the disks were atrophic.

Dr. Graeme M. Hammond suggested that the blindness in the second case shown by Dr. Leszynsky might have been due to wood alcohol poisoning.

Dr. Leszynsky replied that in wood alcohol poisoning the condition of the eyes was one of retrobulbar neuritis and not of choked disk, and furthermore, that the blindness in the former class of cases came on very rapidly.

Dr. L. Pierce Clark said he recently saw a case quite similar to those shown by Dr. Leszynsky, and in his case the patient volunteered the statement that she had been using different sorts of bleaching hair dyes to great excess, and to these she was inclined to attribute her loss of sight. There was in this case a papillitis, followed by atrophy.

Dr. Leszynsky said that no one could make the differential diagnosis between tumor and basilar meningitis by the condition of the optic nerve or the presence of choked disk. He recently saw a case of syphilitic meningitis where the retina was filled with hemorrhages and a high degree of choked disk was preesnt.

Tubercle of the Cerebellum.—Specimen shown by Dr. I. Abrahamson. This case was referred to the speaker by Dr. Samuel Lloyd in order to determine the advisability of an operation. The patient was a male, five years old. His family history was negative. Two years ago he had whooping cough, and about that time began to complain of pain in the head, which his father thought was due to a blow. The pain was always referred to the back of the head, and continued for about a year. Then the left side of the body suddenly became paralyzed, and this paralysis had persisted. For the past two months there had been projectile vomiting, and for the past month the child had been having three or four convulsions daily. During the convulsions, which lasted from five to fifteen minutes, the child was apparently unconscious. He cried a good deal and complained of pain, usually in the head, but also in other parts of the body when attempts were made to move him. He frequently cried out in his sleep. He had lost considerable weight, and there was a notable increase in the size of the head.

Examination showed that the patient was much emaciated. The head was retracted and flexed to the left, and all attempts at movement elicited a sharp cry of pain. The eyes were turned to the left, and upward and downward movements were impossible. The left pupil was more widely dilated than the right, and there was apparently no light nor accommodation reaction. There was no pain reaction. Attempts to look to the right were accompanied by coarse nystagmoid movements. There was marked choked disk. There was general motor weakness, and exaggeration of the triceps, wrist and knee-kerks on the left side. Plantar reflex was not obtainable. Sensibility and special senses were intact. There was no Kernig sign. The thighs and legs were flexed; the feet extended. All attempts to straighten the legs caused pain. The vomiting from which the child had suffered seemed to bear no relation to the food taken into the stomach. It occurred at any time and without warning, and seemed to cause no special distress. When the child was admitted to the hospital he was in a semi-comatose condition, which persisted up to the time of his death. He could be roused, and would answer simple questions fairly and intelligently. He had only one convulsion while in the hospital; this resembled a general spasmodic condition rather than a clonic one. The case was regarded as one of tubercular meningitis complicating general tuberculosis, and no operation was deemed justifiable.

Report of autopsy: On opening the cranial cavity the skull was found to be very thin, even for a child of six years, although the suture lines were well and firmly united. The skullcap was easily removed and the dura incised. Immediately about eight ounces of clear fluid escaped, although the brain itself had seemed to fill the entire cavity. This might possibly be explained by the collapse of the brain, showing that the fluid had occupied the ventricles and escaped through some opening. No tubercular process was found in the meninges covering the vertex. The brain itself was removed without difficulty. On examining the basilar portion of the dura a number of miliary tubercles were found. A gross examination of the brain showed a nodule, about one and a half inches in diameter, situated in the left cerebellar lobe; it was round and fairly regular in outline, and quite firm in texture. No incision was made either into the brain or the tumor itself, the specimen being preserved intact. No tubercular process was found in the leptomeninges. Examination of the other viscera showed a general miliary tuberculosis.

A Case of Acute Dementia or Mental Stupor Following Illuminating Gas Poisoning.—Presented by Dr. Abrahamson. The patient was a woman. 55 years old, a native of Russia. Her family history was negative; the patient had always enjoyed good health, and had never had any previous mental disturbance. Six weeks ago she arose to prepare breakfast for the family on the gas range. After this she returned to bed, and later was found asphyxiated. There was no suspicion of attempted suicide. She was taken to the Gouverneur Hospital, where oxygen was given and phlebotomy done. She remained comatose for two entire days, when her intelligence slowly returned. There was no resulting paralysis or other symptoms, and she left the hospital in an apparently normal condition. Two weeks ago, however, her intelligence became affected. She would remain in one position for hours without a word or sign, apparently entirely oblivious to her surroundings, evincing no emotion whatsoever and making no complaints. There were no delusions nor hallucinations. She did not resist being moved about, and did things automatically. Her facial expression was apathetic. She did not ask for food, but when it was placed before her and she was urged very strongly she was able to feed herself. She answered one of many questions addressed to her, briefly but to the point. When once outside the house, even a few steps from the door, she lost her way. There were no lamentations, no profanity, no spells of restlessness nor excitement, no breach of ordinary decency, no undue exposure. When strongly urged, she recognized individuals, things and places.

Dr. Clark said that three cases like the one shown by Dr. Abrahamson had been seen at the Vanderbilt Clinic. In one of them in which the course and the symptoms were very similar to the one presented, a diagnosis of paramnesia following illuminating gas poisoning was made. This patient recovered entirely in the course of three or four months. The speaker said he did not think the automatism and stupid state in the case shown were sufficient grounds upon which to base the diagnosis of acute dementia.

Dr. Harlow Brooks said that in fatal cases of illuminating gas poisoning, where the patients had survived four or five days, the autopsy occasionally revealed areas of softening in the anterior lobe of the brain, and sometimes in the striate body.

Dr. Smith Ely Jelliffe said that illuminating gas contained carbon monoxide (CO), and it was known that this substance was an active hemolytic poison; hence there might be actual agglutination of blood in the blood vessels, and the production of functional disturbances, such as were presented in the case shown. These lesions also were allied to those spoken of by Dr. Brooks.

Dr. Charles L. Dana said that some years ago he reported a case of illuminating gas poisoning followed by what was described by him as double personality, which persisted for nearly a year. There was a form of memory disturbance, but the patient could talk intelligently, and was in no sense demented. He had forgotten almost everything concerning his previous life. He did not know his parents nor where he lived, but he was able to carry out a number of common, automatic things, and went about his ordinary duties fairly well. He was also able to take care of himself. Dr. Dana said that a number of observations had been made, particularly by French writers, showing that the poison of illuminating gas had a distinct effect on the memory, and that children who were more or less constantly exposed to air contaminated by this gas were apt to have defective memories. The case shown by Dr. Abrahamson was suggestive more of memorv disturbance than of true dementia.

Dr. Abrahamson, in closing said that according to the statements made by the family, his patient had improved somewhat during the past week. Clinically, the symptoms were those of dementia rather than amnesia, as the patient had never been restless or excited, nor had she had any delusions or hallucinations.

Bilateral Cervical Sympathectomy for the Relief of Epilepsy, with Report of Three Cases.—By Drs. William P. Spratling and Roswell Park.

Dr. B. Onuf thought it worth while to examine the excised portion of the sympathetic nerve in all cases where a sympathectomy was done, although if any pathological changes were found in it, it might be difficult to say whether they were primary or secondary.

Dr. Clark said that the first case reported by Dr. Spratling had been under his care for a time. This patient was the first in line of birth of six children; he was a "blue baby," the labor having been instrumental and extremely difficult. The inflammatory changes found in that case might have been due to trauma, and the speaker thought there was some doubt whether the case was really one of true epilepsy.

Dr. Pearce Bailey thought it was rather remarkable that so little attention had hitherto been given to the cervical sympathetic in connection with these cases. Its importance was shown by the result of the pathological findings in the cases reported by Dr. Spratling.

Dr. Spratling, in closing, said that while there was some doubt in the beginning as to whether the case referred to by Dr. Clark was one of true epilepsy, the patient subsequently developed tvpical grand mal attacks. The speaker said he was still a little skeptical in regard to the use of the knife in general in the treatment of epilepsy, and he was rather doubtful whether the improvement in the cases he had reported would be lasting. He had always inclined to the view that epilepsy was a condition usually beyond the aid of the knife, although in many instances even a simple surgical operation, no matter what, seemed to prove at least temporarily beneficial, perhaps by its effect on the general metabolism. This, if nothing else, might render certain operations justifiable in some cases.

Traumatic Epilepsy in Its Medico-Legal Relations.—By Dr. Arthur Conklin Brush.

Dr. Edward D. Fisher said that Dr. Brush's large personal experience with this class of cases rendered his views on the subject of value. The speaker thought the fact was fairly well established, both by clinical observation and animal experimentation, that direct injury to the brain could cause epilepsy even in the absence of any hereditary predisposition.

Dr. Spratling said he entirely agreed with Dr. Fisher. While the occurrence of true epilepsy as the result of a trauma, and independent of any hereditary taint or auto-intoxication, was rare, such cases had come under his observation.

PHILADELPHIA NEUROLOGICAL SOCIETY.

December 27, 1904.

The President, Dr. C. S. Potts, in the Chair.

Dr. Ralph Pemberton exhibited for Dr. Chas. K. Mills a case of Rhizomelic Spondylosis.

Dr. S. D. Ludlum exhibited a case of Spastic Ataxic Paraplegia Developing after Childbirth.

Dr. Spiller said that Dr. Ludlum had reported the case in detail, but there were one or two remarks he wished to emphasize. When this woman first came to the Polyclinic Hospital she was extremely ataxic, but she has improved very markedly. He thought there was undoubtedly a connection between childbirth and her present condition. What her condition was twelve years ago it would be hard to say. Her second child was born without any symptoms following. After the birth of her third child there followed numbness in one lower limb like a pressure palsy, although it was not from pressure. He thought the diagnosis rested between anemic changes in the spinal cord and disseminated sclerosis.

Dr. Pickett stated that at the Philadelphia Hospital about a year ago there was a woman with puerperal confusion, who, in the wards, developed a myelitis, becoming completely paraplegic. She survived, and is now able to walk a little. It seems rational to ally such a case and Dr. Ludlum's likewise with the combined degeneration of Putnam and Taylor due to toxemia; and this alliance might be regarded as a parallel to the well-known connection of pernicious anemia with the puerperium.

In closing the discussion on his case, Dr. Ludlum stated that it did not seem to him that there was enough anemia to account for the changes. The red cells numbered 5,000,000, the whites 10,000 and the hemaglobin 80 per cent. He did not think that indicated sufficient anemia to account for the condition.

A Case of Senile Neuritis and a Case of Hemianesthesia, Hemiataxia Hemiastereognosis, Hemiathetosis and Hemianopsia, Due to a Sudden Cerebral Lesion.—These cases were exhibited by Dr. J. W. McConnell.

Dr. Mills said that he had had the opportunity of seeing the second case in consultation with Dr. McConnell, and the conditions then were as now, only more marked. He thought the case was probably an example of a subcortical lesion. It looked to him like a case where the lesion was in the thalamus and optic radiations. He also thought it possible that the lesion might be in the most posterior part of the internal capsule and optic radiations.

Dr. Burr cited a case he had had at the Philadelphia Hospital last winter of a man who presented symptoms like these, with the exception of the hemianopsia. Just before death he had hemianesthesia, slight palsy, marked ataxia on the left side, and athetoid movements. At the time of the stroke there was palsy of the left side, unconsciousness and anesthesia, the palsy passing off, the other symptoms remaining. At autopsy there was found a softening in the optic thalamus, but the internal capsule had escaped. He also mentioned the case of a woman, now at Blockley, who was in his wards, who had had hemiplegia several years ago. In her case the hemiplegia passed away almost entirely, there being, however, a remnant of palsy left. She has marked ataxia in the left knee, marked anesthesia and athetoid movements, and he believed the case to be one like Dr. McConnell's case, although there was no hemianopsia.

Dr. Spiller said he had had two cases like this, except that hemianopsia was not present. In one, at autopsy the lesion was found to be in the parietal lobe. In both cases there was motor weakness. He stated that in Dr. McConnell's case he thought the lesion could be only in the optic thalamus. He called attention to the fact that this symptom-complex has been attracting much attention, especially among the French. Some call this the syndrome of Dejerine.

Dr. Weisenburg said that he had had the opportunity of seeing this case the second day after the hemorrhage occurred, and at that time the woman had a distinct Babinski reflex, and had weakness of the left upper limb, and he therefore thought the case was one of hemi-paresis at that time. He had also studied a somewhat similar case in the nervous wards of the Philadelphia Hospital. This patient, a man, had hemiplegia. hemiataxia and hemiastereognosis, but he had no disturbance of sensation. On the side of the hemiplegia the patient had rather irregular rotary movements of the upper limb, these movements being coarse, while in Dr. McConnell's case they were slow, rotary and athetoid in character.

In regard to the Babinski reflex, Dr. McConnell said he had tried to elicit this sign before Dr. Weisenburg saw the case, but could not do so. He tried again on the following day, and often since then, but with the same result.

Dr. C. S. Potts reported on a case of Traumatic Cervical Hematomyelia. (To be reported in this JOURNAL.)

Dr. Weisenburg said he had studied this case before the man died, and was present at the autopsy. A transverse depression between the fourth and sixth cervical segments was found at the autopsy. Opposite the fifth and sixth cervical vertebræ was much blood, but there was no displacement of the vertebræ. One interesting point about this case was the state of the pupillary light reflex, which was preserved up to the day before death, when it was absent. Reichardt recently found in an examination of twenty-five or thirty cases of tabes that where the light reflex was absent there was an area of degeneration in the second and third cervical segments. In cases similar to the one under discussion Reichardt stated we could expect an absence of the pupillary reflex only on the last day, and this was so in the case of Dr. Potts. An examination of the second and third cervical segments, however, failed to show any areas of degeneration in any part of the sections, both by the Weigert and the Marchi methods. He thought Dr. Potts' case proves that the center of the biceps tendon reflex is in the fifth cervical segment.

Dr. C. D. Camp read a paper on the pathology of tabes, and another on fibrous nodules of the pia.

Dr. A. R. Allen read a paper on annular degeneration of the spinal cord.

Dr. T. H. Weisenburg read a paper on pseudo-bulbar palsy.

Periscope

ARCHIV FUER PSYCHIATRIE UND NERVENKRANKHEITEN

(Vol. 39, 1904, Part 1.)

1. Clinical Contributions to the Question of Melancholia. O. Kölpin.
2. The Value of the Surgical Treatment of Neuroses and Psychoses. C. Hermkes.
3. Contribution to Knowledge of Endogenous Spinal Cord Fibers in Man. Robert Bing.
4. Angiospastic Gangrene. Hogo Strauss.
5. Contribution to Syphilis of the Brain and the Hypophysis, and to the Differential Diagnosis Between Tuberculosis and Syphilis of the Central Nervous System. Kufs.
6. On the Influence of Anemia upon the Irritability of the White Substance of the Central Nervous System. U. Scheven.
7. Two Cases of Dementia Paralytica with Brain Syphilis. Rentsch.
8. The Myelinization of the So-called Zone of Common Sensibility and the Olfactory and Optic Radiations of Man. Hösel.
9. History and Critique of the So-called Psychic Compulsive States. Warda Wolfgang.
10. Autointoxication Psychoses. E. Meyer.
11. The Condition of the Spinal Cord in Argyll-Robertson Pupil. M. Reichardt.

1. *Melancholia.*—Kölpin discusses the question of melancholia on the basis of eighteen carefully described cases of various forms of depression with special reference to Kraepelin's views. The author lays special stress upon the carefully observed clinical course of his cases, and considers this of more importance than his theoretical discussion. He is in general inclined to take issue with Kraepelin's standpoint, although he recognizes the service which he has rendered in calling attention to the close association between certain melancholic and maniacal states. The matter is discussed on theoretical grounds at considerable length, with the general conclusion that the maniacal conditions may be very considerably differentiated provided sufficiently painstaking observations of the clinical course of the disease are made.

2. *Surgery in Neuroses and Psychoses.*—Hermkes discusses the indications for surgical interference in persons suffering from neuroses and psychoses, taking the conservative ground that surgical interference is only justified in those cases in which a definite indication is apparent. Special care must be taken in determining the objective grounds for operation, and the most painstaking attention must be given to the psychic condition of the patient. In general, only such cases should be operated upon which, did a psychosis not exist, would demand interference, and even in such cases it is well to defer the operation, if possible, until the psychosis disappears.

3. *Endogenous Spinal Cord Fibers.*—Bing makes a careful study of endogenous fibers in the human spinal cord on the basis of a case of acute poliomyelitis of unusual severity in a child of four months. The autopsy showed a hematogenous myelitis in the distribution of the anterior spinal arteries, with parallelism between the parenchymatous and interstitial alterations. Since the lesions were essentially confined to the gray substance, extending in the lumbar region into the posterior horns, and death resulted at such a time that use could be made of the Marchi method, the case was

peculiarly adapted to the study of the endogenous fibers. Some of the conclusions reached are as follows: The direct cerebellar tract begins in the region of the transition from the second to the third lumbar segment; Gowers' tract appears to begin one segment below the beginning of the cerebellar tract; endogenous tracts are found in the area of the crossed and uncrossed pyramidal tracts; the endogenous ground bundle fibers of the ventral and lateral tracts are ascending in their course; the endogenous dorsal tract fibers are for the most part descending in their course.

4. *Angiospastic Gangrene.*—Strauss concludes from his study of angiospastic gangrene, usually known as Raynaud's disease, that it is a form of so-called spontaneous gangrene due to spasm of vessels, that, inasmuch as it often appears on the one side, often both sides, but rarely sharply symmetrical, the term symmetrical gangrene is unfortunate. It is, therefore, suggested that the term angiospastic gangrene be substituted, which also avoids confusion with other forms of spontaneous gangrene. The gangrene itself is essentially the final consequence of a lack of tissue nutrition following spastic ischemia or spastic cyanosis, except that in the latter case autointoxication occurs as a favoring cause of the gangrene. There is, therefore, no ground to regard Raynaud's disease as a trophoneurosis. It is rather a pure angioneurosis occurring at times as an independent disease, and at times on the basis of other diseases of the nervous system.

5. *Syphilis of Brain.*—Kufs reports in detail a case of syphilis of the cerebral convexity combined with gumma of the hypophysis and gummata in a cirrhotic liver. The object in publishing the case is to draw attention to certain peculiar pathological findings with the histological investigation of the diseased organs, which the author regards as offering noteworthy results, particularly in relation to differential diagnosis between tuberculosis and syphilis. The clinical history of a woman of 47 is given in detail, followed by an exhaustive discussion of the pathological findings. The frequent difficulty between the diagnosis of these two conditions is discussed at some length with reference to the literature. The article is an example of the exhaustive work over a single case characteristic of the German mind.

6. *Anemia and Cerebral Irritability.*—As a result of experimental work Scheven comes to the conclusion that the assumption is justified that the white matter loses its irritability for the induced current in the same way as the gray in conditions of anemia, but that the final word is by no means given in the question through the foregoing statement.

7. *Dementia Paralytica and Syphilis.*—Two cases are reported of dementia paralytica combined with brain syphilis. In the first case there was disease of the basal arteries confined to a part of the vertebral artery, the basilar and the two anterior cerebrals, a typical arteritis gummosa. In conjunction with these lesions there was a typical diffuse degenerative alteration in the brain cortex, to be regarded as a pure primary disease of the type of paralytic dementia. In the second case a gummatous tumor was found at the base of the brain, and in this case also the hemispheres showed the typical lesions of dementia paralytica. The interest of these cases lies in the definite association of primary degenerative lesions and typical syphilitic processes, the pseudo-paralysis syphilitica of Jolly.

8. *Myelinization of Tracts.*—Hösel's study is purely anatomical in character, and is of such a detailed nature that it does not lend itself to a brief abstract. The paper is of value to those who are studying fiber tracts of the brain, and is confirmatory of observations made by others.

9. *Compulsive States.*—This article is continued in the next number of the *Archiv.* Its abstract is, therefore, deferred.

10. *Autointoxication Psychoses.*—On the basis of several cases Meyer discusses the justification of the assumption of autointoxication in the production of symptoms. He concludes that it is not possible on the clinical side to speak of specific autointoxications so far as the mental

disturbances are concerned, although one may reach a juster conclusion by consideration of the physical symptoms as well. Furthermore, Meyer is of the definite opinion that the anatomical alterations observed in these cases do not admit of the assumption of a specific cause. In his cases of psychosis with autointoxication the anatomical changes were strikingly like those first described by Bonhoeffer in delirium tremens. Nothing more is to be said than that there is a general anatomical expression of the autointoxication to be observed post-mortem.

11. *Spinal Cord and Argyll-Robertson Pupil.*—Reichardt, in discussing the relation of the spinal cord to the Argyll-Robertson pupil, sets himself the following questions: Is there a characteristic degeneration in the upper cervical cord in cases of tabetic and paralytic immobility of the pupil? Under what circumstances may the spinal cord appear normal in cases of immobile pupil? Under what circumstances may this pupillary phenomenon appear in other diseases of the spinal cord? As a result of his very careful anatomical and historical discussion of the subject, he concludes that the cause of the immobility of the pupil is not to be sought in the ciliary ganglion, but that the assumption must be made that the peripheral reflex arc remains intact and that a higher arc suffers degeneration in the causation of the failure of the pupil to respond to light. He does not, however, deny that certain cells of the ciliary ganglion may be degenerative in consequence of the condition which brings about the Argyll-Robertson pupil.

E. W. Taylor.

NEUROLOGISCHES CENTRALBLATT

(Vol. 23, 1904, No. 15, August 1,)

1. Concerning Magneto-Electric and Sinusoidal Currents from an Electro-Diagnostic Standpoint. M. Bernhardt.
2. Familiar Symmetrical Monodactylia. E. Schultze.
3. Korsakoff's Symptom Complex after Brain Injury. E. Meyer.
4. What Is the Condition of the Pupil in Typical Pupillary Rigidity? L. Bach.

1. *Sinusoidal Currents.*—Continued Article.

2 *Monodactylia.*—Schultze records a remarkable case of a boy of nineteen who had only one finger on each hand and one toe on each foot. These were symmetrical. The other portions of the body were normal. The X-ray photographs show that there are only seven bones in the wrist and only three metacarpal bones in the left foot and two in the right. One sister, his mother and his mother's father have similar conditions.

3. *Korsakoff's Syndrome.*—Meyer records a case of injury to the head which was followed by the typical symptoms of Korsakoff's psychosis.

4. *Pupillary Rigidity.*—By ordinary pupillary rigidity is meant loss of light reaction and not of convergence, although this interpretation is placed upon it by some authors. Bach examined the condition of the pupils in a large number of cases of pupillary rigidity and found the pupils narrower than normal. Convergence reaction is normal, except perhaps that the pupils contract slower than usually. At times the condition of ordinary pupillary rigidity may pass into one of absolute rigidity, in which the pupils are wider than normal and do not respond to convergence or light. The latter condition may occur first and pass into the former, but the conditions may appear independently. In ordinary pupillary rigidity Bach, according to his and Meyer's experiments, places the lesion either in their inhibitory centers in the medulla oblongata or in the uppermost part of the spinal cord; and in the condition of absolute rigidity he places the lesion, first, peripherally, and secondly, either in the ciliary ganglion or in the oculomotor nucleus.

(Vol. 23, 1904, No. 16, August 16.)

1. Remarks upon Alterations in the Cerebellum in Tabes Dorsalis.—C. Weigert.

2. Concerning the Dissociation of Superficial and Deep Pain Sensation in Cerebral Hemiplegia.—LIEPMANN.
3. Concerning "Mechanismus and Vitalismus."—H. HAENEL.
4. Concerning Magneto-Electric and Sinusoidal Currents from Electro-Diagnostic Standpoint.—M. BERNHARDT.

1. *Cerebellum and Tabes Dorsalis.*—Weigert has observed for years in those cases of tabes dorsalis in which he was able to stain by the neurogliar method alterations in the molecular layer of the cerebellum. These consist in an increase and thickening of the neurogliar fibers. The same observation is true in paretics.

2. *Dissociation of Sensation in Hemiplegia.*—Liepmann records a case of an idiot with a left hemiplegia, who, when operated upon for a boil, did not manifest any discomfort while the deeper tissues were cut, but did when the skin was incised.

3. Continued article.

4. *Sinusoidal Currents.*—Bernhardt, in an exhaustive article, considers the relative value of sinusoidal currents in peripheral and spinal diseases, and comes to the conclusion that our present knowledge fails to show definitely the therapeutic value of such currents.

(Vol. 23, 1904, No. 17, September 1.)

1. Hypotonia and Hypertonia in the Same Patient. Z. BYCHOWSKI.
2. The Etiology of a Thyroid Gland Disappearance in Cretins and in Myxedema. DR. BAYON.
3. The Pupillary Changes on Lateral Deviation of the Eyeball. A. MARIANA.
4. A New Apparatus for Photographing Pupillary Movements. J. PILTZ.

1. *Hypotonia and Hypertonia.*—The author records a case of paralysis agitans in which the upper extremities were rigid and hypertonic, the facies was typical of paralysis agitans, as was also the tremor, while in the lower extremities the patient had lancinating pains, inco-ordination and hypotonia.

2. *Thyroid Gland Disappearance.*—Bayon insists upon the same etiology for sporadic and endemic Cretins, the disappearance of a thyroid gland. In sporadic cases the Cretin is born without a thyroid gland, while in the endemic form the disease of the thyroid gland will come on early in life, due perhaps to some inter-current, infectious disease.

3. *Pupillary Changes.*—Mariana discusses the causes of a contraction of the pupils in convergence, and records two interesting cases in which, on lateral deviation to the right, the left pupil contracted and the right did not. He obtained these phenomena in three cases. No definite cause as yet can be given.

4. Continued article.

(September 16, 1904, No. 18.)

1. Gluteal Reflexes. W. v. BECHTEREW.
2. The Blood Serum Treatment of Epilepsy. H. GERHARTZ, Jr.
3. An Apparatus for Determining the Brain Volume.—F. REICH.
4. The Present Status of the Question of Heredity in Neuro- and Psycho-Pathology. E. HÄHNLE.
5. A New Apparatus for Photographing Pupillary Changes. J. PILTZ.

1. *Gluteal Reflex.*—Bechterew describes aother reflex.

2. *Epilepsy.*—The treatment of epilepsy by serum injection was carried out according to Ceni's method. This depends upon two factors which are in the blood of epileptics, one a toxic and the other an antitoxic element. (Ceni's article was abstracted in a previous number of the JOURNAL OF NERVOUS AND MENTAL DISEASE.) The author treated two cases for about eighteen months, but did not obtain any results. He believes, however, that the treatment should be carried further.

3. The article is unsuited for abstracting.

4. Continued article.

5. Piltz describes a new apparatus for photographing pupillary changes. The readers are referred to the original.

(October 1, 1904, No. 19.)

1. The Present Status of the Question of Heredity in Neuro- and Psycho-Pathology. E. Hähnle.

In a long and exhaustive article the author discusses the question of heredity in nervous and mental diseases, and seems to agree with Sioli's views regarding the importance of hereditary transmission. He advances the following conclusions:

(*a*) The principal cause of a large number of nervous and mental diseases in about one-half of the total number of patients is an inherited neuro- and psychopathologic disposition.

(*b*) The knowledge gained by neuro- and psychopathology argues for a hereditary disposition.

(*c*) A definite exposition of the etiological factor is at present impossible.

(*d*) A differential diagnosis between hereditary and acquired diseases is at present impossible.

(*e*) The hypothesis that a diseased hereditary disposition may not be eradicated in some cases is probably an error, for such instances exist.

(October 16, 1904, No. 20.)

1 A Contribution to the Question of Choked Disc. W. Uhthoff.

2. Microscopical Changes in a Case of Esophageal Carcinoma in the Vagus Nucleus. Preliminary Contribution. C. Hudovernig.

3. A Case of Abducens Paralysis of the Extremities after Injury to the Skull. E. Bloch.

1. *Choked Disc.*—Uhthoff refers to the work he has done either by himself or with others, and records 240 cases of choked disc. Of these all but four had a definite diagnosis; one hundred and thirty-four of these cases were due to brain tumor and twenty-seven due to syphilis, and so on. He believes that the percentage of cases due to syphilis was greater than his number indicates. He doubts the occurrence of primary optic neuritis due to infectious causes.

2. *Vagus Nucleus Changes.*—Hudovering, in a case of carcinoma of the esophagus, found alterations in the nerve cells of the ninth and tenth nucleus. He considered these alterations due to "reaction at distance." By this means he attempts to map out anatomically the relation between certain organs and their central innervation.

3. *Abducens Paralysis.*—Bloch records an interesting case in which, following a fracture of the base of the skull, there was a paresis of the external rectus muscle of the right side, and also a paresis of the right upper and lower limbs. He assumes that the involvement of the external rectus muscle was due to a hemorrhagic exudation.

(November 1, 190, No. 21.

1. The Symptomatology of Paralysis Agitans. L. Bruns.

2. A Contribution to the Etiology of Cervical Ribs. H. Levi.

1. *Paralysis Agitans.*—Bruns records some unusual symptoms of this disease. He has seen in a number of cases an abnormal tendency for the secretion of saliva and agrees with Oppenheim, who first described these symptoms, that it is a bulbar manifestation; but he goes farther and records four cases in which there were typical bulbar symptoms, such as difficulty in talking, swallowing, and an increased secretion of saliva. There was, however, no atrophy of the lips and tongue. Bruns assumes that the lesions in these cases are not nuclear, but that we have here a condition analogous to pseudo-bulbar palsy. He also discusses trophic symptoms,

atrophy of the limbs and the conjunction of multiple sclerosis with paralysis agitans and paralysis agitans with tabes dorsalis.

2. *Cervical Ribs.*—Levi refers to a statement of Oppenheim that in bilateral cervical ribs there is a tendency to syringomyelia. He records an interesting case of bilateral cervical ribs in which there were accompanying symptoms of multiple sclerosis. Both diseases are congenital in origin.

(November 16, 1904, No. 22.)

1. A Case of Syphilitic Meningitis of the Base and Convexity. K. Schaeffer.
2. A Further Contribution to the Determination of the Brain Volume. N. P. Petzy.
3. A Contribution to the Staining Methods of Nerve Fibers in the Central Nervous System. A. Kozowsky.

1. *Syhilitic Meningitis.*—Schaffer records an interesting case of a patient who had syphilis. The following points were of interest: In the macroscopical examination there was a complete atrophy of the right frontal lobe. This extended to the precentral convolution. The atrophy also involved the tip of the right temporal lobes, and there was absence of the right olfactory bulb. The patient had Jacksonian convulsions and disturbance of sensation, ataxia and impulsive laughing. The first three symptoms can be explained by the involvement of the right central convolutions. Concerning the impulsive laughing Schaffer agrees with Brissaud that this is probably due to a destruction of the "Faisceau psychique," whose course is between the frontal lobe and the anterior limb of the internal capsule. Concerning the secondary degenerations there was a degeneration of the anterior limb of the internal capsule and the median bundle of the peduncle, this agreeing with the findings of other authors.

2. *Brain Volume.*—The author discusses Reich's previous contribution upon the measuring of the brain volume, and advances an apparatus of his own.

3. The article is unsuited for abstracting.

Weisenburg.

MONATSSCHRIFT FUER PSYCHIATRIE UND NEUROLOGIE

(Vol. 15, 1904, No. 6, June.)

1. Contribution to the Knowledge of the Spinal Cord and the Pyramidal Tracts of the Tripa Europaea. J. Draseke.
2. The Symptomatology of the Cerebellum (Cerebellar Hemiataxia and Its Origin. L. Mann.
3. Headache in Maniacal Depressive Insanity. Diehl.
4. Electro-Diagnostical Investigations with the Help of the Condensator Method. Th. Ziehen.
5. Isolated Hallucinations Developing in Epileptics. Lachmund.
6. The Annual Assembly of the German Society for Psychiatry at Göttingen from April 25 to 27, 1904. Weber and Vogt.
7. Scientific Assembly of the Physicians of the St. Petersburg Psychiatricial and Neurological Clinic, May 16, Oct. 3 and 24, 1902, and Jan. 23, Feb. 27 and March 27, 1903. F. Giese.

1. *Pyramidal Tracts in Talpa.*—Draseke has made a careful study of the spinal cord of the mole. He calls particular attention to a small mass of gray matter which is situated in the anterior white columns. Traced in an upward direction this area acquires medullated fibers, which can finally be seen to belong to the pyramidal columns of the medulla. It appears, therefore, that this area represents the pyramidal columns which in the lower animals, such as the mole and the hedgehog, are characterized by the fact that the medullary substance is very difficult to stain, and in the lower part of the axis cylinders appears to lose this capacity entirely. The observation confirms Obersteiner's belief that the pyramidal columns rep-

resent a later phase of development than some other parts of the spinal cord.

2. *Cerebellar Hemiataxia.*—Mann reports a case characterized by hemiataxia without hemianopsia. A diagnosis of cerebellar tumor was made and both cerebellar hemispheres were carefully explored, with negative results. The patient died, and at the autopsy a large tumor of the left occipital lobe was found, which had pressed upon the left cerebellar hemisphere, but apparently had not involved the optic tract. No changes were found in the right cerebellar hemisphere, although Babinski's reflex was present on the left hemiatactic side. He concludes with some speculation upon the physiology of the cerebellum. He regards it as the mechanism which lies outside our consciousness, but fulfils the impulses which are transmitted from the consciousness to produce voluntary movements, and that it is connected with the muscles by means of centripetal tracts.

3. *Headache in Manic Depressive Insanity.*—Diehl reports two interesting cases of headache which was merely a symptom of mental disease. The first patient, a woman, attracted his attention by the manifest excitement under which she was laboring at the time of his first visit, inquiry developed the fact that she was in the habit of taking large quantities of hypnotics and analgesics. Careful treatment of the mental condition and the removal of drugs sufficed for a cure. The second patient was, properly speaking, a case of morphinism, in which the indication for treatment was the intense headache The patient was treated for the habit, partially cured, and upon her return to her family indulged to excess in social dissipations, whereupon she developed an attack of mania which required prolonged treatment for its improvement.

4. *Electro-Diagnosis.*—The results of a series of investigations with the condensator method were not so much to aid in the diagnosis of certain forms of paralysis as to yield a more exact and profound knowledge of the pathological processes that underlie them. The results hitherto have been scanty, but certain results have been obtained and expressed in figures that are at least curious. These are obtained by the application of a rather complicated formula, for the discussion of which we must refer to the original paper.

5. *Hallucinations in Epilepsy.*—Lachmund reports three cases of epilepsy in which hallucinations occurred, sometimes during an attack, sometimes during an interval or as a sequel of an attack. In the first case, a man of 28, who showed distinct signs of dementia, the hallucinations were visual in character. During the attacks the field of vision was narrowed, but at times during the existence of the hallucinations it was fully normal. The second case, a woman of 38 years, with neuropathic heredity, had had an attack of insanity during each puerperium. The hallucinations were aural in type, and usually occurred after the attack and lasted for several days. While they existed there were no other apparent mental symptoms. The third case, a man of 37, had two or three severe attacks every year. There was progressive dementia. After the attacks he had delirium and terrifying hallucinations. During these hallucinations he was not confused. Bromide intoxication was excluded in this case.

6. The following papers were read at the annual meeting of the German Society for Psychiatry held at Göttingen, April 25 to 27, 1904.

Neuropathology and Psychiatry. By Fürstner.

The Health and Educational Institutions in Göttingen, with Especial Consideration of the Sanitorium Rasemühle. By Cramer.

The Ganser Symptom. By Henneberg.

The Classification and Nomenclature of the Psychoses with Reference to the Requirements of Medical Examination. By Hoche.

The Dietetic Treatment of Epilepsy. By Alt.

The Relation Between the Chemical Constitution and Hypnotic Action; A New Group of Hypnotics. By Schultze.

Psychiatry and Pedagogics. By **Wanke**.
The Korsakoff Symptom-Complex in Its Relation to Various Forms of Disease. By Bonhoeffer.
Hysterical Insanity. By **Raecke**.
The Value and Significance of Cytodiagnosis for Mental and Nervous Disease. By **Siemerling**.
Again Concerning the Marriage of Persons Previously Insane. **Schüle**.
The Relation of the Brain in Situs Viscerum Transversus. By **Weygandt**.
The Physiology of the Patellar Reflex. By **Scheven**.
Experimental Psychopathology. By **Sommer**.
A Demonstration of Microscopical Preparations of a Rare Case of Malformation of the Spinal Cord. By. **Westphal**.
Demonstrations of the Cytc-Architecture of the Cerebral Cortex with Especial Reference to the Histological Localization in Various Mammals. By **Brodmann**.
Serial Sections Through the Brain of a Case of Apraxia. **Liepmann**.

7. At the scientific assembly of physicians of the St. Petersburg Clinic for Psychiatry and Neurology the following papers were read:

The Ascending and Descending Degenerations of the Spinal Cord in Compression Myelitis of the Lower Cervical and Upper Dorsal Portions. By **Schukowsky**.
The Central Associations of the Brain Nerves in Human Beings. By **Troschin**.
The Cortical Centers for the Secretion of Sweat. By **Gribojedow**.
Hallucinatory Psychosis as a Result of Disease of the Organs of Hearing, and the Influence of Attention upon the Localization of Hallucinatory Objects. By v **Bechterew**.
The Influence of Opocerebrin upon the Irritability of the Cerebral Cortex By **Ossipow**.
A Study of the Conduction Tracts of the Cerebellum, According to the Method of Embryonal Development. By **Tkatschenko**.
A Case of Brain Tumor with Occult Occurrences. **Iwanow**.
A Case of Stiffness of the Spinal Column. By **Troschin**.
Supraorbital Non-Progressive Atrophy. By v. **Bechterew**.
Akathisie. By v. **Bechterew**.
Local Apraxia. By v **Bechterew**.
The Singulum. By **Troschin**.

(Vol. 16, 1904, No. 1, July.)

1. Experimental Studies upon the Pathogenesis of Mental Diseases. H **Berger**.
2. Spaces Between the Individual Teeth: An Early Diagnostic and Hitherto Little Known Sign of Acromegaly. W. **Graves**.
3. Recent Experiences in the Mental Disturbances Following Poisoning with Sulphide of Carbon. F. **Quensel**.

1. *Pathogenesis of Mental Diseases.*—Berger has attempted to produce manifestations of insanity in dogs. First he employed subcutaneous injections of serum of a case of dementia precox, but the quantities required were so large and the results so slight that he abandoned the method. He then tried the intracerebral injection of the same serum, perforating the skull under general anesthesia, and later injecting the serum, usually employing from 10 to 20 ccm., and making the injection over the occipital lobe at the level of the ectolateral gyrus. Altogether 25 animals were injected. Of these, 7 animals were infected by the operation in spite of all precautions. In one animal death occurred shortly after the injection, in status epilepticus. In 4 animals severe injury to the brain was produced by the injection. In 13 animals neither epilepsy, infection or death occurred as a result of the injection. Of these, 5 animals showed symptoms

that might be ascribed to the toxic action of the serum in the form of peculiar chronic twitchings, that disappeared in from one to three hours. According to Berger, this indicated that there is some toxic substance in the blood of patients suffering from dementia precox that is capable of irritating the motor cortex of the dog's brain. The animals injected with serum from cases of hallucinatory insanity, circulatory insanity, melancholia and post luetic dementia showed no symptoms. The paper is still unfinished.

3. *The Teeth in Acromegaly.*—Graves describes a case of juvenile acromegaly which commenced in a girl at about the age of 12 years, and was observed by him when she was 14½ years old. The hands and feet had not enlarged, the chin was not prominent, but it was possible to determine that the lower teeth had become more widely separated. This condition was not found in other members of the same family. Graves discusses the crania progenea which occurs in acromegaly, cretinism, in individuals who in early childhood have had severe attacks of smallpox, in degenerates, and in apparently healthy individuals. He calls attention to the fact that not only does the lower jaw project in this condition, but it also becomes broadened, giving rise to the separation of the teeth. He reports a second case in a woman 38 years of age, who had had symptoms for about nine years. The separation of the teeth was more marked than the prominence of the jaw. The third case was a man of 43, in whom the separation of the teeth was one of the earliest symptoms. Graves discusses the literature of this condition, and concludes that in no other disease is broadening of the jaw and the consequent separation of the teeth so constant as in acromegaly.

3. *Sulphide of Carbon Poisoning.*—Quensel reports three cases of poisoning with sulphide of carbon. The paper is still unfinished.

(Ergänzungsheft, Vol. 16, 1904.)

1. Acute Fatal Psychoses. L. W. Weber.
2. The Psychical Diseases of Animals. H. Dexler.
3. A Case of Bilateral Disappearance of the Muscles of the Calves. A. Knapp.
4. Casuistic Contribution to the Chapter on Asthenic Paralysis. W. Sterling.
5. Pupil Testing and Pupil Reaction. J. Donath.

1. *Acute Fatal Psychoses.*—In contrast to acute delirium there occurs a symptom-complex beginning acutely either in health or in the course of chronic psychoses, which is characterized by depression and the catatonic symptom-complex. It is either impossible to arouse the patient or else he shows automatic response to commands. Death occurs without any apparent impairment of nutrition or the presence of any infectious disease, excepting possibly an agonal pneumonia. Weber reports 5 cases of this condition, all characterized by almost complete lack of objective symptoms, either in the form of paralysis or cortical irritation. There was no fever, but apparently defective circulation, because the hands and feet were cyanosed, although the heart's action appeared to be good. The chief psychical symptom was inhibition. The patients sometimes showed a considerable degree of anxiety. Nutrition was well maintained in all, but death occurred in all cases after a relatively short interval. In case 1 death occurred 46 days after the first symptoms; case 2, 13 days; case 3, 4 months; case 4, 11 days; and in case 5, 16 days. Three of the cases were associated, respectively, with epilepsy, general paresis and senility. Two of the cases Weber regards as pure functional psychoses. In none of the cases were any significant changes found in the central nervous system. Weber does not regard this symptom-complex as the expression of a disease process uniform either etiologically or anatomically. There is probably often some

primary psychical defect. The predisposing causes are, among others, injuries and neuropathic heredity. The vascular changes resemble in some respects those of acute delirium.

2. *Psychical Disease in Animals.*—Dexler, after discussing the literature regarding the psychical manifestations in animals, reports a series of cases that he was able to observe, and upon which he made careful autopsies. The microscopical examination in all cases showed that an inflammatory condition of the central nervous system was present, usually in the form of a disseminated meningo-encephalitis, sometimes with involvement of the spinal cord. The clinical histories and descriptions of the microscopical examinations are given at great length. All dogs had previously suffered with distemper. Pick, who examined them, regarded the dogs as suffering from deficient consciousness, but not insanity. The symptoms may be described as follows: General depression of all the special senses, of the muscular sense and the sense of position, preservation of all the vegetative functions. There was a marked tendency to fall asleep in unusual places or in uncomfortable positions. The movements were clumsy and the animals often fell; they were stuporous; the pupillary and tendon reflexes were preserved. Choked disc did not appear. The reflex of attention, that is, the licking of the nose, was present in all the animals. In conclusion Dexler states that none of the three animals can be regarded strictly as insane or demented, but all suffered from a peculiar kind of encephalitis which produced a general depression of the sensoria and various forms of focal lesion. Therefore, there is no reason to believe that they indicated the existence of genuine insanity in the lower animals.

3. *Bilateral Loss of Calf Muscles.*—A man of 25 years received a blow from a heavy stone on the lower part of the back. Immediately he felt extreme weakness in the legs, and difficulty in the passage of urine and feces. In a short time, however, he was able to resume work. Knapp examined him at the age of 45, and found complete disappearance of the calf muscles, absence of the electrical reaction in the calf, disappearance of the Achilles tendon reflex, and inability to rise on the toes. There was also moderate hypesthesia for all forms of sensation on the feet and legs. He regards the case as due to tearing of some of the spinal nerves at their exit from the sacral foramen, probably those arising from the first and second sacral segments.

4. *Asthenic Paralysis.*—Sterling reports six cases of asthenic paralysis. In the first case psychical disturbances exerted a profound influence upon the course of the disease. In the second case the electrical irritability of the facial nerve was diminished. The fourth case commenced after an operation for abscess in the mouth, and presented certain peculiarities in the electrical reaction; that is to say, that the difference in the force between the first and second contractions was greater than between any consecutive subsequent contractions. The fifth case was interesting because the symptoms appeared to involve chiefly the lavator palpebræ superioris The sixth case is doubtful. Sterling discusses the etiology briefly. One of the patients died, but there is no record of autopsy.

5. *Pupillary Reaction.*—Donath contributes a summary of the recent literature of the pupillary reflexes.

J. Sailer.

AMERICAN JOURNAL OF INSANITY

(Vol. 61, 1904, No. 2.)

1. Hydrotherapeutics. George T. Tuttle.
2. Feigned Insanity; Malingering Revealed by Use of Ether. Charles G. Wagner.
3. Hospital and Asylum-Trained Nurses. C. P. Bancroft.

4. Observation Wards and Hospitals. E. STANLEY ABBOTT.
5. Sarcoma of the Orbital Periosteum. E. M. GREEN.
6. Mental State in Cretinism. E. E. MAYER.
7. Sensation and Motion. FREDERICK C. GESSNER.
8. Types of Alcoholic Insanity. B. H. W. MITCHELL.
9. A Case of Moral Insanity with Repeated Homicides and Incendiarism. and Late Development of Delusions. HENRY R. STEDMAN.
10. Some Metabolism Studies with Special Reference to Mental Disorders. OTTO FOLIN.

1. *Hydrotherapeutics.*—The author gives some account of his experience with hydrotherapy at the McLean Hospital since 1899. In some selected cases ergographic curves and blood pressures were taken before and after bath treatment (generally hot air, followed by douches both hot and cold), blood counts were made, and studies in metabolism were carried out by Dr. Otto Folin. The ergograph did not give very positive results; if anything, there was slight loss of energy. The pulse rate was raised and the blood pressure lowered by heat, the opposite effect being produced by cold. There appeared to be a temporary increase in the number of blood corpuscles in blood taken from the ear after cold applications, which the author attributes to narrowing of the surface vessels. Working on a fixed diet, it did not appear that metabolism was more active under bath treatment. On the whole, the author can come to no very definite conclusions, though he thinks that the bath treatment, through its effect upon the circulation, may improve nutrition, and its suggestive effect is not to be undervalued.

2. *Feigned Insanity*—The case of a condemned murderer who the defense claimed was suffering from lateral sclerosis and demented, and who simulated with sufficient skill to raise for some time a doubt in the minds of the experts of an examining commission. It occurred to Dr. Wagner to put him under the influence of ether, pushed to complete anesthesia. Under this, in the stage of excitement the patient talked quite naturally, and when partially out of its influence he walked with a natural gait, exposing the simulation. He later confessed that he had been malingering, and was duly executed.

3. *Hospital and Asylum Trained Nurses.*—The author emphatically affirms that training schools in asylums have come to stay, but recognizing how serious a handicap in the matter of securing the proper kind of attendants, is lack of sufficient practical training in actual nursing of medical and surgical cases, he makes a strong plea, first, for the establishment in every large asylum of a real hospital building, at which the pupil nurses are to serve in turn; and second, as far as practicable the affiliation with and interchange of pupils between asylums and general hospitals, and also, where possible, district nursing associations.

4. *Observation Wards and Hospitals.*—A plea for the establishment of detention and observation wards for cases presenting mental symptoms, either connected with general hospitals or separate, in all cities large enough to create a need for them, with some account of the workings of such wards in New York, Albany, Boston and Philadelphia.

5. *Sarcoma of the Orbital Periosteum.*—The case of a negress, 28 years of age, presenting the following features: a malignant growth involving the periosteum of the orbit, violent maniacal outbreaks, frequent convulsive seizures; following the removal of the growth and the eye, restoration to the normal mental condition, disappearance of the convulsions, and so far no recurrence of the growth.

6. *Mental Condition in Cretinism.*—A discussion of the mental state of cretins, and its differences from that in arrested mental development from other causes, on the basis of two personally observed cases, and a review of the literature of the subject.

7. *Sensation and Motion.*—An abstract discussion, containing, however, a suggestion that in visual perception "the parenchymes of the nerve cells engaged in the sensory act of seeing do at the same time perform a motor act by originating vibrations commensurate to those of light," the description of experiments to prove which by a suitable apparatus being promised later.

8. *Types of Alcoholic Insanity.*—Among 1,129 patients admitted to the Danvers Insane Hospital during five years there were 148 cases of alcoholic insanity. On these the author bases a clinical study, grouping them under the following divisions: 1, Delirium tremens, 41 cases; 2, alcoholic hallucinosis, acute 33 cases, subacute 25 cases; 3, alcoholic delusional insanity, 34 cases; and 4, alcoholic dementia, 13 cases. From each of these groups he selects some typical examples whose histories he gives. His conclusions are as follows:

"I. Heredity of insanity or intemperance is common, and has an unfavorable influence on the course of the disease.

"II. The persistence of tactile and olfactory hallucinations in cases of subacute hallucinations affects the prognosis unfavorably.

"III. Somatic and grandiose delusions of a changed personality in alcoholic delusional insanity indicate a chronic course, with a probable unfavorable termination.

"IV. A history of periodical habits of drinking is more common in delirium tremens and hallucinosis, and of daily drinking in the delusional type of dementia.

"V. The free use of distilled liquors is noted almost without exception in all cases.

"VI. Hallucinosis but rarely develops from an attack of delirium tremens, but is often preceded by one or more attacks.

"VII. Alcoholic hallucinosis often serves as the basis for elaboration of the delusional system seen in the delusional type."

9. *A Case of Moral Insanity.*—The interesting medico-legal case of "J. T.," a professional nurse, 45 years old, who confessed to poisoning 31 persons, in four of these cases, the fact being duly proven by chemical analysis, and in 16 more being substantiated by strong corroborative evidence. The author, with Dr. Jelly and Dr. Quinby, having been appointed by agreement between the prosecution and the defense a commission to examine this woman, unearthed a strong heredity of insanity and intemperance, with a life history of defect in moral and ethical feeling, which, however, did not take the form of sexual obliquity or indulgence in alcohol or narcotics. All evidence went to show that the patient had full knowledge of the wrongfulness of her acts, but she evinced a levity and entire lack of feeling of regret or remorse, and there was no apparent motive for her crimes. The commission hence regarded the woman as defective and mentally irresponsible, but as there were no symptoms to attach the case to any clinical type, viewed it as one of moral insanity. Their findings were accepted, and the woman committed for life to the Taunton Hospital. The justice of the course taken was fully vindicated by the sequel, for within two years after her commitment the patient had developed delusions of persecution—especially of poisoning—with marked mental deterioration, refused food and had faded away to a shadow of her former self.

10. *Some Metabolism Studies with Special Reference to Mental Disorders.* By Otto Folin (Concluded from Vol 60, No. 4).—When one considers the expense, time and labor involved in the work whose results are here represented, and of which it is impossible to give more than the barest outline, one cannot but feel that it is a subject for felicitation that we have in this country, at any rate, one institution whose managers are sufficiently imbued with the scientific spirit to authorize and encourage a work requiring so high a degree of special training,

involving such an enormous amount of labor, calling for so much in the way of special apparatus and skilled assistance, and withal presenting such a doubtful prospect for the attainment of results of a sufficiently practical character to appeal to the lay mind.

In developing his plan of work the author at the start had to contend with inadequacy and unreliability of chemical methods, and was forced in most instances to elaborate them for himself. These methods he describes here and elsewhere. He points out that there are on record few complete analyses of urine—from the same person—and outlines his method, which was, after putting his patients upon a fixed diet, to make daily analyses of their urines. His diet consists of the following daily ration: Whole milk, 500 c.c.; cream (18 to 22 per cent. fat), 300 c.c.; eggs (white and yolk), 450 grms.; sugar, 20 grms.; malted milk, 200 grms.; salt, 6 grs.; water enough to make 2 liters. After two days of this diet the total daily urine was collected and subjected to a complete analysis, the following constituents being determined: Phosphoric acid, sulphuric acid, chlorine, ethereal sulphates, nitrogen, urea, ammonia, kreatinin and uric acid, together with acidity mineral and organic, specific gravity, and quantity in 24 hours. The food was analyzed from time to time, but the analysis of the feces was not attempted. To secure as near as possible a normal standard seven healthy employees, all males, were put upon the above diet and their urines analyzed in the regular way. The results showed that while there was a tendency towards uniformity, there were also variations in the different constituents, the most striking variation being in the quantity of urine passed in 24 hours. In insane patients on the same diet, however, these variations tended to extend themselves through much wider limits. Examining the relationships between the different constituents he found the nitrogen-sulphate ratio remarkably constant in normal cases, and in most of the insane. Even more constant was the urea-ammonia-nitrogen ratio. The kreatinin-nitrogen ratio and the uric acid-nitrogen ratios varied within narrow limits in normal persons. The nitrogen-phosphate and sulphate-phosphate ratios varied far more among the patients than among the normal individuals. An extended series of feeding experiments in a case of general paresis showed remarkable variations in a number of urinary constituents, the most striking being the nitrogen-sulphate ratio, which showed the sulphate figure constantly below that found in normal cases. Seven other cases of general paresis showed the same tendency to low nitrogen-sulphate ratio. These findings, the author thinks, indicate very strongly that altered metabolism is a prominent feature in general paresis. In this connection there is mentioned a remarkable case which presented for a long period a daily alternation in condition, one day being quiet and nearly normal, the next showing great restlessness and deterioration. On bad or "nervous" days the phosphate elimination was much above that on good days, the nitrogen-phosphate ratio varying accordingly. Attempts to modify this condition by diet, however gave no positive results. This case was long thought to be one of manic-depressive insanity, but an autopsy made later in another institution proved it to be one of general paresis. The results of the examinations in the above cases are exhibited in tabular form, as are those obtained from 36 other patients, male and female, suffering from different forms of mental disease. As to these latter cases, all that the author feels able to say is that individual variations in the urinary constituents seem to be more common than in normal individuals. Summarizing, he thinks that these are the most complete and extensive metabolism experiments upon the insane on record, and asks "What do they teach?" He answers, from a positive point of view, little that is tangible, except that they contain a strong suggestion that general paresis may be associated at one stage or another with demonstrable disorder of metabolism. From a negative point of view they prove the untrustworthiness of those metabolism experiments which report characteristic "increase" or "diminution" of any of the above-mentioned urinary constituents as associated with any particular mental disorder. From a general physiological point of view, he believes the data contained in his tables I to XLVII of value, as giving the complete composition of a large number of urines.

MISCELLANY

ACUTE MENINGITIS. W. T. Councilman (Journal A. M. A., April 1).

The author describes the conditions of acute meningitis with more special reference to that form produced by the *Diplococcus intracellularis meningitis,* which he believes is constantly occurring in a sporadic form, aside from the not infrequent epidemic aggravations. The infecting organism, he states, is one of low vitality and incapable of a purely saprophytic existence. The statistics fail to give any adequate idea of the frequency of the infection in ordinary years. His experience, however, leads him to believe that with rare exceptions all cases of primary meningitis are due to this micro-organism, and that it would be impossible without sporadic infection to bridge over the intervals between the epidemics. It is possible, too, that the germ may even inhabit the normal mucous membranes of the nose, for example, as has been shown in a few cases where it produces a rhinitis, and infection of the meninges may take place through the lymphatics or by continuity of surface. We can only explain the epidemics of the disease, he says, either by an increase of virulence of the diplococcus or by a decrease in the resistance of the tissues. The underlying causes of epidemics are unknown, and even atmospheric conditions cannot be excluded. He discusses to some extent the relations of meningitis to pneumonia, as shown by the Massachusetts health statistics, and illustrates with a chart. Primary meningitis from the pneumococcus or staphylococcus is rare; secondary types are not so infrequent. The paper concludes with a description of the pathologic conditions in acute meningeal disease.

TREATMENT OF IDIOPATHIC EPILEPSY. D. R. Brower (Journal A. M. A., March 25).

Brower says that spontaneous cure of this disease is possible, and mentions one of several cases in his observation. He calls attention to the necessity of more care as to the prevention of the trouble, especially in infants suffering from convulsions. The proper treatment and environment in these cases may prevent the later development of the disease. The treatment of the individual convulsions is also important, and the aura may afford a warning that enables the patient to abort the attack. He advises the carrying by epileptics of nitrite of amyl pearls for this purpose. Other methods may also be effective in special cases. In epilepsy there is an autointoxication, usually of gastro-inestinal origin, and the diet should be carefully regulated. These patients are usually very hearty eaters, and it is advisable to restrict the diet in quantity, to regulate periods of eating and to insure thorough mastication and digestion. Intestinal elimination must also be attended to, and for intestinal antisepsis he finds salol combined with phytolacca often very useful. For combating the nervous irritability the bromids are most useful, and he prefers the sodium salt. Their overuse, however, is dangerous, and Brower refers much of the existing epileptic insanity to this cause. The dose should seldom exceed sixty grains daily in plenty of water after eating, and he sometimes adds fl. ext. of *Solanum carolinense* in ½ to 2 dram doses to the the bromid mixture. Strychnia is also a valuable remedy for meeting the circulatory and vasomotor defect, and he specially recommends fluid extract of *Adonis vernalis.* Cerebral sclerosis calls for alteratives. In conclusion, he insists on the importance of allowing plenty of time, at least five years after disappearance of symptoms, before claiming a cure of epilepsy.

Book Reviews

Uber Familienähnlichkeiten an den Grosshirnfurchen des Menschen. Von Dozent Dr. J. P. Karplus, Assistent am physiologischen Institut in Wein. pp. 58 and plates. Leipsig und Wein. 1905.

Karplus has attempted in this monograph, reprinted from the investigations of the Neurological Institute at Vienna, to study the central nervous system with respect to family resemblances. Apart from the somewhat vague possibilities which such a research would be likely to develop, difficulty was naturally experienced in the accumulation of material. This difficulty was, however, in a measure overcome through the courtesy of various professors in Vienna who were able to place at the author's disposal a considerable amount of suitable material. The final research includes twenty-one groups of central nervous systems, of which sixteen had two members, four three members, and one five members. A painstaking study of the convolutions and grosser aspects of these nervous systems was made, and comparisons instituted in the hope of being able to answer the question whether there in an hereditary transmittion of brain fissures. The conclusions reached from so small a material for study are naturally not far reaching, a fact which the author willingly confesses, expressing, however, the expectation that possibly a new method of investigation of the nervous system has thereby been opened. The monograph is beautifully illustrated by a large number of plates, naturally a far safer method of demonstrating the points at issue than a mere descriptive text. The central nervous systems studied are arranged by families in table form, a method which lends itself to ready reference. The author in general has evidently expended a large amount of labor in the development of a subject, which on its face offers peculiar difficulties and small hope of immediate value. This service should be acknowledged. No doubt the external configuration of the brain still offers possibilities for further study, but it is certainly a far less fertile field than the study of the brain cortex and the course of its fibers.

E. W. Taylor.

Webster's International Dictionary.

The G. and C. Merriam Co. of Springfield, Mass., have accomplished a very worthy deed in getting out an up-to-date edition of that standard favorite, Webster's International Dictionary. This new edition contains a Gazeteer and Biographical Dictionary, brought up to modern standards, and altogether the volume is a commendable presentation of an invaluable reference work, whether for office, school or home.

Studies in the Psychology of Sex—Sexual Selection in Man. I, Touch. II, Smell. III, Hearing. IV, Vision. By Havelock Ellis. F. A. Davis Company, Philadelphia.

The present volume is the fourth from the pen of this erudite and facile writer dealing with the general subject of the psychology of sex. In it is discussed the subject of sexual selection in man from the view-point of the four special senses: touch, smell, hearing and vision.

At the outset the author takes occasion to refute the erroneous principle set forth by Darwin in the "Descent of Man" that supposes sexual selection to depend upon an æsthetic sense that recognizes beauty as a desideratum. Mr. Ellis says very aptly with reference to this conception: "When we look at these phenomena in their broadest biological aspects, love is only to a limited extent a response to beauty; to a greater extent beauty is simply a name for the complexus of stimuli which most adequately arouses love."

Following the introduction the subiect of sexual selection is considered with relation to the four special senses mentioned. Throughout the work the author maintains a broad biological standpoint. The part the different senses play is appreciated and discussed on the basis of their origin from the primitive sense of touch, and the several points taken up are illustrated by a wealth of examples culled from the writings of scientists, travelers and physicians of all ages.

The chapters on vision are the longest and most comprehensive, naturally, as this sense plays such an important part in sexual selection in man, and being so widely removed from its prototype, touch, the problems connected with it are much more obscure. The author observes a catholicity of spirit in dealing with these obscure phases of sexual psychology, and his conclusions are reached tentatively and with a full appreciation of the often inadequate evidence upon which they are founded.

The volume, as a whole, may be said to be a careful, painstaking and learned exposition of the problems of sex of which it treats, and can be confidently recommended to the intelligent seeker after light in this most obscure domain of psychology as being quite the best modern work in English. WHITE.

LECTURES ON CLINICAL PSYCHIATRY. By DR. EMIL KRAEPELIN, Professor of Psychiatry in the University of Munich. Revised and Edited by Thomas Johnstone, M.D., Edin., M.R.C.P., London. William Wood and Company.

The readers of this JOURNAL do not need reminding that the study of psychiatry has been taken up with tremendous energy during the past twenty years. It has remained for most a difficult subject, and does so still because of the necessary faults in the psychological substructure. For as long as phenomena of the mind are to be sorted, classified and arranged by the organ yielding the very phenomena it would bring into orderly groupings, then considerable variation in matters of interpretation is inevitable.

And so one finds it to-day in the writings of alienists who make any pretense to a modern knowledge of brain physiology from the psychological side.

Kraepelin has been one of the leaders in modern psychiatry, and as one reads the lectures here printed in most acceptable English dress the reason for his leadership is made very apparent, for founded on the best psychological data of Wundt and his followers, Kraepelin has added a superstructure of clear clinical pictures that rival those of the foremost masters in neurological medicine.

The work is preëminently a clinical one. Picture after picture is revealed, each yielding up valuable material for alienist and general practitioner alike. For the psychiatrist no extended review is needed; it can be truly said, however, that this work is a very desirable one, and makes an excellent explanatory volume to Kraepelin's larger studies. JELLIFFE.

News and Notes

THE OPENING OF GLENWOOD.—On May 15 this new home for epileptics will be opened. Special rates will be offered to early applicants. From the standpoints of location, training of those in charge and accommodations offered Glenwood promises to be an ideal institution for the treatment and care of epileptics.

A New Neurological Society.—A number of physicians met in St. Louis on April 20, 1905, and voted to inaugurate a neurological society in that city, to be designated The St. Louis Neurological Society. Those present were Drs. M. A. Bliss, Given Campbell, Chas. G. Chaddock, Frank R. Fry, W. W. Graves, H. W. Hermann, M. W. Hoge and Sidney I. Schwab. Dr. Fry was chosen president and Dr. Given Campbell secretary.

PROGRAM OF THE THIRTY-FIRST ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION,

JUNE 1, 2 AND 3, 1905.

Presidential Address: The Importance in Clinical Diagnosis of Paralysis of Associated Movements of the Eyeballs, Especially of Upward and Downward Associated Movements, by Dr. William G. Spiller; the Relation of Certain Persistent Emotional States to Insanity, by Dr. Theodore H. Kellogg; A Fatal Case of Neurasthenia, by Dr. Theodore Diller; Insanity in the Aged, by Dr. Charles W. Burr; Psychasthenia, Its Clinical Entity, Illustrated by a Case, by Dr. Sidney I. Schwab; A Study of Pathological Gait, by Dr. Joseph Sailer; Multiple Sclerosis: Four Cases, with Autopsy; Consideration of Differential Diagnosis, by Dr. Edward Wyllis Taylor; The Relation of the Cerebral and Olfactory Regions of the Brain, by Dr. Burt G. Wilder; Two Cases of Cervical Rib, with Operation, by Dr. Hugh T. Patrick; A Contribution to the Acute Polioencephalitis Superior (Wernicke), by Dr. J. Ramsay Hunt; Disease of the Cerebral Venous System, by Dr. D. J. McCarthy; A Study of Two Brain Tumors of Endothelial Origin; One a Multiple Cylindroma, One a Perithelioma, by Dr. Herman C. Goldinier and Dr. H. H. Carey; The Results of Operations for the Removal of Cerebral Tumors, by Dr. Philip Coombs Knapp; The Operability of Intracranial Growths, by Dr. George L. Walton and Dr. W. E. Paul; Post-operative Asthenia, by Dr. S. Weir Mitchell; The Motor Area of the Human Cerebrum as Determined by Cortical Localization, with a Discussion of the Surgery of the Motor Region, by Dr. Charles K. Mills and Dr. C. H. Frazier; Cerebellar Tumors with Reference to Diagnosis and Surgical Treatment, by Dr. B. Sachs; Sarcoma of the Cerebellum with Unusual Symptomatology, by Dr. F. X. Dercum; Concerning Operative Intervention for the Intracranial Hemorrhages of the New-born, by Dr. Harvey Cushing; The Progressive Spinal Atrophies; A Study of the Etiology and Grouping of Fifty Cases, with Report of a Case with Autopsy, by Dr. Charles L. Dana; Ascending Transverse Myelitis with Osseous Anchylosis and Growth of the Vertebræ, and Extreme Anterior Curvature, by Dr. Graeme M. Hammond; Carcinoma of the Nervous System, by Dr. William G. Spiller and Dr. Theodore H. Weisenburg; Observations of the Temperature in Cerebral Hemorrhage and Cerebral Embolism, by Dr. William N. Bullard; Palates of the Feeble-Minded, by Dr. Walter Channing and Dr. Clark Wissler; Paraplegia from Fracture of the First, Second and Third Dorsal Vertebaæ, by Dr. Guy Hinsdale; A Case of Brown-Sequard Paralysis Due to a Fall upon the Head, Operation, Autopsy, by Dr. Wm. C. Krauss; A Case of Epilepsy Associated with Graves' Disease and Total Alopecia, by Dr. Frank K. Hallock; Hypesthesia or Hypalgesia and their Significance in Functional Nervous Disturbances, by Dr. Edward B. Angell; Sarcoma of the Spinal Cord with Dissociated Sensory Loss, by Dr. F. X. Dercum; Hysterical Stigmata Caused by Organic Brain Lesions, by Dr. Herman H. Hoppe; The Mental Symptoms of Cerebral Tumor, by Dr. Philip Coombs Knapp; A Case of Double Consciousness, by Dr. Richard Dewey; Exhaustion Paralysis of Cortical Origin Due to Arteriosclerosis, by Dr. D. J. McCarthy; A Case of Internal Hydrocephalus with Autopsy, by Dr. Theodore Diller; The Chronic Progressive Softening of the Brain, by Dr. J. Ramsey Hunt; Autopsy in a Case of Adiposis Dolorosa, by Dr. F. X. Dercum.

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THE
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Original Articles

DEMENTIA PRÆCOX.[1]

By B. SACHS, M.D.,
OF NEW YORK.

There has been no end of trouble in attempting a proper classification of mental diseases. At different periods and in different nations varying systems have been devised based either upon a resemblance in symptoms, upon the acuteness or chronicity of development or upon a common etiology. Any special subdivision can be justified only if it helps us to recognize distinct clinical entities, if it helps us to differentiate one form of disease from another; each of the diseases so differentiated should have a cause and a symptomatology all its own. The morbid conditions underlying it should in some sense be peculiar to it, but I realize that it would be useless at the present time to insist upon a special morbid pathology of each mental disease. We have a right, however, to insist when a special form of physical derangement is presented for our consideration, that such a form shall be easily recognized and that the recognition of it shall prove to be a distinct advance upon previous conceptions of it and its allied forms of disease.

The insanities of early life and of the adolescent period have a special claim upon the attention not only of alienists but of all medical men. Society has a right to expect of us that we should, if possible, bring about segregation of these tainted individuals and prevent (if it is within our power) the increase through marriage of these more or less defective individuals. For the last decades we have been accustomed to diagnosticate the pubescent insanities hebephrenia, katatonia, the delusional and confusional forms of early life, as well as primary dementia, acute manias, melancholias, and the hypochondriacal forms of adolescence. We are to abandon most of these if not all of them and are to substitute dementia præcox. What do we gain by this substitution? In opening the discussion upon this subject I wish to elicit an expression of opinion on this,—one of the pressing questions of the day. The collective experience of a large number alone can decide where the truth lies.

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[1] Read at the meeting of the American Neurological Association, Sept. 15, 16, 17, 1904, to open the discussion on Dementia præcox.

The French claim that the idea of dementia præcox was entertained by Esquirol, but that later on it was lost sight of in the purely degenerative forms of mental disease described by Morel and Magnan. There is no doubt, however, that the clinical delineation of hebephrenia by Hecker, of katatonia by Kahlbaum, led up to the conception of dementia præcox as promulgated by Kraepelin and his followers. A careful study of Kraepelin's writings, more particularly the last edition of his excellent text-book, shows that dementia præcox is to denote a "psychosis of early life characterized by a peculiar and progressive deterioration of all the intellectual faculties, ending in dementia." Before this deterioration sets in the patient may have appeared to be tolerably normal or he may have passed through periods of excitement or depression, of marked confusion; he may have been subject to delirium, to delusions, to hallucinations; he may have exhibited special forms of stereotyped utterances and actions (the constant repetition of definite phrases and of definite, often eccentric movements), or he may have been in a condition of stupor, of katatonia or of negativism (a condition in which the patient's will seems totally paralyzed). The dementia is a general one embracing all the faculties but is never complete, in which respect it is supposed to differ from dementia paralytica and from senile dementia.

It is evident that this definition is broad enough to embrace almost every form of psychic derangement in the young. From my own experience in private practice and in the various institutions with which I have been connected, I can state with some confidence that there is a great temptation to diagnosticate a vast majority of the cases of mental diseases in youthful individuals as cases of dementia præcox. This would be simple enough if it were altogether true or justifiable: the age of the individual would of itself postulate the diagnosis. It is against these tendencies of the day that I consider it fair to enter a strong protest. Many alienists have evidently exceeded the intentions of Kraepelin himself. He distinguishes between the hebephrenic, the katatonic and the paranoiac forms of dementia præcox: The only feature which they have in common is a supposed termination in the condition of dementia. Kraepelin states, however, that in seventeen per cent. of the cases, dementia never sets in and that in others there are long periods of remission during which term the patient enjoys perfect health.

In former days we were well content to make the diagnosis of an acute attack of mania, of melancholia, of hypochondriasis. We reconized special conditions of hebephrenia and of katatonia, and we were contented to add that if the attacks were frequently repeated or if the condition did not yield to treatment for a prolonged period of time, and if there was a distinct hereditary taint, a dementia might ensue. Is there any gain, in pushing the idea of a dementia into the foreground, in putting the seal of ultimate mental deterioration upon these cases of youthful insanities? There could have been no more unfortunate selection of a term, but I do not object to terms nearly as much as I do to the ideas which they convey. We often call tabes dorsalis locomotor ataxia, although there may never have been any ataxia from beginning to end of the disease. But in this present day conception of dementia præcox, the very term implies a grave and, I believe, a far too grave prognosis in many of the cases so labelled. I wish, however, to be distinctly understood when I grant that there are definite cases which fit in admirably with the description as given by Kraepelin. It will not do in this discussion to give a complete record of cases, but I wish to instance a few and I take them only from my private records covering a period of years so as to give some idea of cases which, to my mind, are properly enough termed cases of dementia præcox, and others to which the term might be applied by the followers of Kraepelin, but in which recovery has either set in or in which, after a lapse of years, the mental condition would not warrant a suspicion of a dementia or even of a general deterioration.

Let me call attention first to the case of a southern gentleman, now twenty-eight years of age, who has been under my observation for at least three years. He comes of a tainted though distinctly bright family. His father committed suicide in a fit of depression, and an uncle of his was under my care about ten years ago suffering from marked hypochondriasis from which he recovered. The patient got along well enough at school; he was distinctly eccentric in his choice of comrades. Testimony of his friends was to the effect that he was a lad of average intellect. At the age of nineteen he began to study law, but was never able to pass his examinations. A few years ago he passed through a state of excitement and was advised to travel abroad. At the end of a year he returned to the study of law, but was chagrined to find that he could not compete with his fellow students. He broke down utterly, became depressed and imagined that his family were planning to prevent his success as a lawyer. In a period of intense excitement he was removed to a private hospital, where for a whole year he was a mere automaton exhibiting not the slightest signs of intelligence, amusing himself by making clicking sounds, and in reply to all questions addressed to him his stereotyped phrase was, "My brother John is a lawyer." Unexpectedly and almost over night his mind cleared up, he began to talk intelligently and took an interest in political and business affairs. He showed a lack of judgment and weakness of his reasoning powers, however, for he has not at any time inquired into his financial affairs (he is a man of wealth), and although he knows that a committee of his person has been appointed, has never requested that such committee be removed. In such a case as this the diagnosis of dementia præcox and possibly of the paranoid form may be allowed to stand.

I have in mind another case in which a well-known alienist made the diagnosis of a dementia præcox many years ago. It was the case of a professional man who was entirely unequal to the demands made upon him by his university studies and who for years failed to obtain a degree. At the end he succeeded, and although he never entered into the practice of his profession, he takes a great and intelligent interest in all professional work. For many years he has not shown the slightest eccentricities and to all intents and purposes is in good mental health. It is not necessary to go into the full details of this history, but it is evident that even if there be some mental inferiority, it is a distinct injustice to such a patient as this, to make a diagnosis of dementia, whether it be a dementia præcox or any other form of dementia.

Many years ago I was asked to see a young student who had under a great strain of an emotional character completely broken down and become depressed and somewhat emotional for a short period of time. He developed delusions of persecution, passed through a period of marked mental enfeeblement, but after a lapse of six months or thereabouts recovered sufficiently to return to his family from the asylum to which he had been committed. This was before the days in which Kraepelin's writings had exerted their influence, but if the young man had been examined at the present day and had presented the symptoms of ten years and more ago, the diagnosis of dementia præcox would no doubt be insisted upon. Yet this same young man has become one of the prominent lawyers in this city and has been one of the leading spirits in every reform movement. The diagnosis of dementia præcox in such conditions would have been manifestly unjust. The list of such experiences could be easily increased, but for the purposes of this discussion it is sufficient to insist that even young subjects may pass through temporary periods of depression or excitement, through periods of transitory mental enfeeblement, and may for a time show what would appear to be a progressive deterioration of mind, but often enough, and fortunately, the process is arrested and the patient regains full mental vigor. In such conditions it is far preferable to speak in the terms of the old nomenclature, to refer to attacks of transitory youthful insanity than to put upon the individual at once the stigma of *dementia* præcox.

To anyone who has witnessed the changes in psychiatric nomenclature during the last score of years, it will be evident that 'though the diseases remain the same, our designation of them undergoes marked variations. The diagnosis of paranoia which was at one time so ready to hand, appears to be a far less frequent one. Paranoia and the primary insanities are now largely designated as paranoid forms of dementia præcox. The present writer, however, is willing to confess that in spite of the protest against the universal acceptance of this term, the understanding of the youthful insanities has been considerably advanced by the introduction of the term dementia præcox and much further good is to be expected of its use if only a little more discrimination is shown between the various forms of mental derangement occurring in youthful individuals.

For the purpose of this discussion, which I have the honor to introduce, I would lay especial stress upon the following points: There are unquestionably many cases that correspond accurately to the types described by Kraepelin and his followers. This is particularly true of the earlier forms of mental derangement occurring in members of families in which there is a very marked psychic taint. But even in such individuals many years may pass before appreciable dementia sets in. The term should be carefully restricted to such cases in which mental deterioration at an early stage of the disease is clearly recognizable and should be carefully considered and possibly avoided in those cases in which a dementia may possibly be developed in the far distant future. Making the diagnosis of dementia præcox puts the stamp of an incurable malady upon persons who may be sufficiently alert to be useful to themselves and others for a long period of years and in that sense does them distinct injustice. There seems to be little gain in grouping widely different conditions under one heading simply because the individuals so afflicted are in the first third of life. The older plan of clinical subdivision is more commendable, and the tendency to dementia should be insisted upon only when there is reason to think that a deterioration is certain to develop at a relatively early period.

A CASE OF TRAUMATIC CERVICAL HEMATOMYELIA AND COMPLETE DIVISION OF THE CORD, WITH PROBABLE DISLOCATION OF THE FIFTH CERVICAL VERTEBRA. REMARKS ON THE LOCATION OF THE CENTER FOR THE BICEPS REFLEX.[1]

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Summary.—A white male aged 56 years, who after a fall down stairs was immediately rendered unconscious for about twelve hours. On recovering from which, with the exception of some delirium several days later, mentality was good. Complete motor and sensory paralysis involving the legs and trunk and partially involving the arms. Loss of all tendon reflexes excepting the biceps, which, however, was lost temporarily on the right side. Preservation of the cilio-spinal, cremasteric and plantar reflexes, the latter disappearing shorting before death. No Babinski phenomenon. Death on the ninth day. At the autopsy was found a possible slight displacement of the fifth cervical vetebra; marked compression of the cord, so as to practically divide it between the sixth and seventh cervical segments. Hemorrhage into the gray matter extending from the eighth cervical up to and partially destroying the right posterior horn of the fifth segment. There was also a fracture of the right side of the skull, with extensive extradural hemorrhage compressing the motor area, which, excepting the early period of unconsciousness, had caused no symptoms.

History.—A. C., aged 56 years, was admitted to the Nervous Wards of the Philadelphia General Hospital on September 11, 1904. His temperature being 101 degrees, pulse 90, and respiration 35 to the minute. He stated that about twenty-four hours before admission, after drinking several glasses of ale, he had fallen down stairs, and was immediately rendered unconscious, remaining so for about twelve hours. When he recovered consciousness it was found that the legs and arms were paralyzed. Examination September 12, about 36 hours after the receipt of the injury, showed a well-developed man, who answered questions intelligently and stated nothing of note concerning his previous or family history. The temperature, pulse and respiration were normal. He lay in bed on his back with the arms elevated about 15 degrees from the body, the forearms flexed on the arm and somewhat supinated, and the fingers flexed.

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[1] From the Neuropathological Laboratory of the University of Pennsylvania and from the Philadelphia General Hospital. Read before the Philadelphia Neurological Society, Dec. 27, 1904.

The pupils were equal and responded to light and accommodation. There was no paralysis of any of the cranial nerves. He could elevate each arm to a right angle with the trunk, and they could be rotated. Flexion of both forearms could be performed, but not with normal strength, the forearms could be slightly supinated. The abdomen was somewhat distended. The legs were completely paralyzed. No atrophy was noticed. Electrical reactions were not taken. The muscles were flaccid.

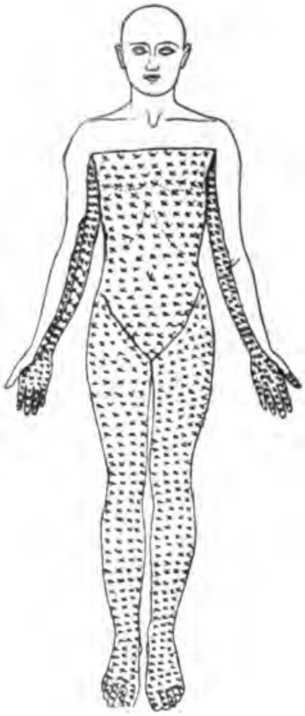

FIG. 1.

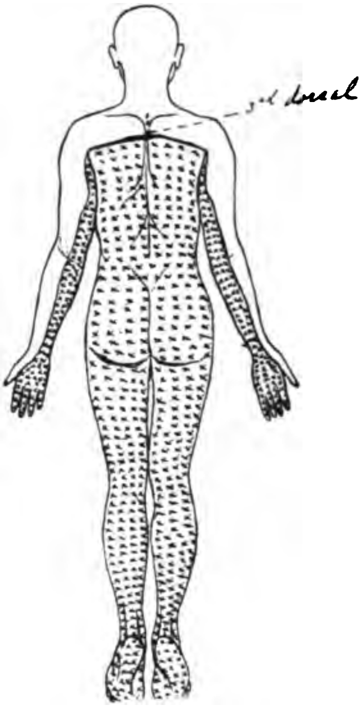

FIG. 2.

The cilio-spinal reflex was present on both sides, the biceps jerk was present and active on both sides, the triceps, wrist, knee and Achilles tendon jerks were absent, there was an active cremaster reflex, and when the soles of the feet were stroked there was slight flexion of the toes. Retention of urine and constipation were present and at times there was slight priapism.

The distribution of the sensory paralysis (tactile, pain and temperature) was as is indicated in Figs. 1 and 2.

The patient complained of pain in the back of the neck, and there was some tenderness in the lower cervical region, but no deformity was visible, either to the eye or in the skiagraph. Bed sores were beginning over the right trochanter and the scapulæ, and the stick of a pin was followed by an area of hyperemia which persisted for some time.

The urine was acid and contained albumin and hyaline and granular casts.

On September 14 it was noted that the patient was delirious, but could be made to answer questions and obey commands. The abdomen was distended and tympanitic. During respiration the abdominal muscles did not contract for several seconds: they then contracted spasmodically one or more times, to be followed by a period of rest as before. The arms could be elevated, and rotated outward further than at the first examination, otherwise there was no change. On September 16 it was found that the toes were neither flexed nor extended when the soles of the feet were irritated. On the right side the biceps jerk was absent, but was very active on the left side. He was delirious at times but answered questions intelligently when spoken to. The peculiar respiratory movements noted on the 14th had disappeared. The left pupil was slightly dilated, but both responded to light and in accommodation. The cilio-spinal reflex could not be obtained. The temperature was 96, pulse 40; on the 17th these again became normal; respiration normal. On Sept. 18 Dr. Weisenburg noted that the patient's face was cyanotic, the left pupil dilated, and the left palpebral fissure slightly narrowed. His voice was weaker, and he could not cough as well, but he could swallow. A slight biceps jerk on the right side was obtained. The patient died on September 19, 1904. The autopsy was made about twenty-four hours after death by Dr. A. G. Ellis, Dr. Weisenburg being present, to whom I am indebted for the following report:—

The scalp in the left frontal and the left parieto-occipital region is ecchymosed. There is a fracture of the skull, starting from a point one-half inch to the right of the median line, at the junction of the parietal with the occipital bone. The line of the fracture is to the left, forward and downward, and ends in the squamous portion of the temporal bone just in front of the ear.

The skull is unusually and uniformly thin, at its thickest portion not measuring more than one-eighth of an inch. Inside of the skull, in the distribution of the left middle meningeal artery there is an extensive extradural hemorrhage. This lies behind the central fissure. The brain, when removed, shows a depression in the region of the hemorrhage, but otherwise nothing is found macroscopically.

The tissues surrounding the upper cervical vertebræ are congested. There is apparently no fracture or dislocation of the cervical vertebræ[2]; at least the vertebræ are not movable and crepitation is not felt. The cervical vertebræ from the seventh to the third inclusive, were removed intact, and show nothing abnormal, except that opposite the fourth and fifth cervical vertebræ there are adherent clots of blood. The upper portion of the third cervical and the second cervical vertebræ were removed piece-meal.

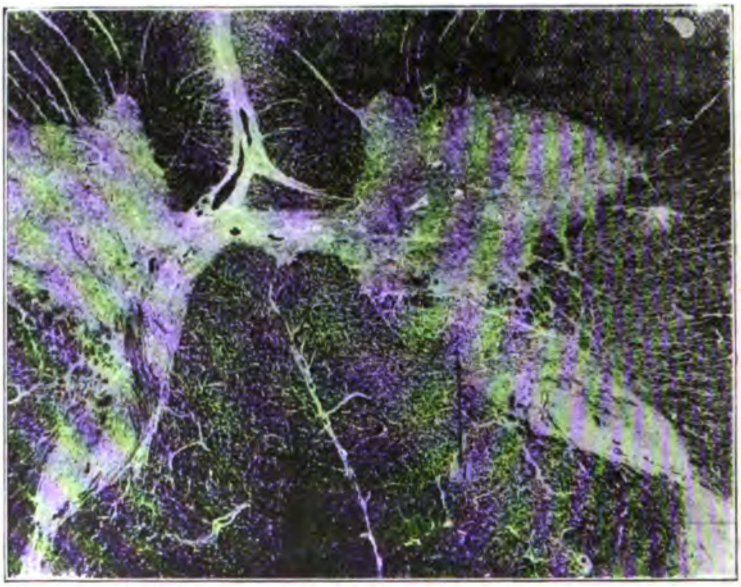

FIG. 3.—Photograph of a section from the fifth cervical segment showing an area of softening and hemorrhage in the right posterior horn (A).

The spinal cord in its upper portion, between the sixth and seventh cervical segments, showed a transverse depression, the cord measuring here about one-eighth of an inch. For an inch both above and below this part the cord was soft to pressure.

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[2] Dr. Ellis thought that the fourth cervical vertebra was displaced slightly backward, but from the location of the compressed area of the cord it is more likely that the fifth had been displaced and then was spontaneously reduced.

Microscopical Examination.—The compression, which is very intense, is between the sixth and seventh cervical segments. Sections were made through the lower part of the fifth cervical segment, which was the utmost limit of the area of softening. The area of compression was not cut, as it was unnecessary and it was not thought advisable to destroy uselessly a valuable gross specimen.

At the level of the second and third cervical segments the spinal cord shows no alteration. There is no ascending or descending degeneration even by the Marchi method, as the patient only lived one week after the accident.

At the level of the fifth cervical segment the right posterior horn is destroyed in its ventral half by softening and hemorrhage. (Fig. 3) The posterior half appears to be normal. Fibers are seen entering the right posterior horn and stopping a little short of the area of softening within the horn. The softening seems to be almost confined to the right posterior horn. This is the only portion of the cord at this level which seems to be pathologically altered, the left posterior horn apparently being normal. It is presumable from a study of the sections that the posterior root supply of the fifth cervical segment must have been impaired on the right side.

Sections taken from the lower part of the fifth cervical segment stained by thionin show considerable alteration of many of the nerve cells. In a number of these the nucleus is displaced to the periphery and the chromophilic elements are disintegrated and appear as fine granules, and some of the cells are swollen. This alteration is seen in the anterior horn of each side.

In the lower part of the eighth cervical or the upper part of the first thoracic segment the cells of the anterior horn are very little diseased.

The spinal cord at the first thoracic segment and below appears to be normal. The twelfth thoracic and the second and fourth lumbar segments are perfectly normal. Special attention has been directed to this part of the cord, because of the loss of all the tendon reflexes.

The most interesting feature of this case is the evidence that it affords of the location of the center for the biceps jerk. This is usually given as the fifth and sixth segments. On the left side in this case the biceps jerk was very active until the death of the patient, while on the right it was active for the first couple of days, then disappeared, to again become feebly present two days later. Microscopic examination of the specimen shows that on the left side the fifth segment is intact, while on the right side the posterior horn is partially destroyed by hemorrhage and softening. (Fig. 3). It seems probable therefore that at first this segment was not involved on the right side, but later became so by the hemorrhage gradually extending upward, at which time the reflex disappeared; then as some absorption took place, the functions of the segment partially returned, and it again became feebly present. As the biceps jerk was very active while the fifth segment was intact, it seems likely that this segment has most to do with the biceps jerk, and that the sixth plays only a subordinate part, if any.

The fact that the eighth cervical and first dorsal segments were intact accounts for the preservation of the cilio-spinal reflex.

While Bastian claimed that both superficial and deep reflexes are abolished in complete transsection of the cord, this case conforms to what has usually been noted by observers, i.e., the cremaster and plantar reflexes were present, it differs however in the fact that the plantar reflex gave the flexor and not the extensor response, which according to Turner is the rule. This possibly might have developed if the patient had lived longer. Attention is called to the fact that there was no lesion in the lumbar cord.

The distribution of the motor paralysis corresponds to the muscles usually regarded as supplied by the affected segments.

The area of sensory paralysis does not correspond to the diagrams of Head, but with the exception of the non-involvement of the thumb is similar to those of Starr and Kocher.

That there was an extensive extradural hemorrhage over the motor area of the left side which gave no symptoms, if we except the period of unconsciousness present immediately after the fall and a possible weakness of the muscles about the angle of the mouth in the right side when the man was admitted, but which had disappeared the next day, is a fact of interest.

Note—Since the foregoing was written Dr. Morton Prince has reported in the JOURNAL OF NERVOUS AND MENTAL DISEASE, Feb., 1903, p. 81, a case in which one side of the cord was severed between the sixth and seventh cervical segments. The area of anæsthesia in his case is similar to that in the above case, i.e., the thumb is not involved, and in this regard differs from the diagrams of Starr, Kocher and Wichman.

THE MEDICAL TREATMENT OF INSANITY.[1]

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The great aim of medical art is the cure of disease, and in fulfilling this there are certain general principles which commend themselves to every medical practitioner, quite apart from the specialized treatment which is applied to the various maladies described in detail in the many text-books upon medicine. These principles are, in short, hygienic, prophylactic, and directly remedial or therapeutic, and they apply to the treatment of insanity with even greater force than they do to diseases in general; for insanity results from neglect of the rules of health and through attempts made to override or to disregard the laws of Nature. In hygienics we have the study of conditions which are of immense importance to the welfare of the whole population, such as those relating to occupations, to the density of the population, to drainage, air and ventilation, to the character of the water and of food, and also those relating to personal and general cleanliness. The neglect of the laws of health deduced from these studies involves a lowered vigor and vitality, a diminished power of resistance to disease, and a general deterioration, if not a degeneration of both mental and bodily energy; the output of work is in consequence diminished, and any given stress approaches nearer the breaking point. Hygienic measures are of so much importance that the State now recognizes the necessity for the individual attention of medical men and others in carrying them out, and public bodies find their highest function in establishing and perfecting conditions favoring a high standard of health among a community.

Prophylactic or preventive treatment implies that there are circumstances, exposure to which tends to impair the general health, either directly or through tendencies derived from parents. Questions relating to alcohol, and to immorality agitate the public mind from time to time in regard to insanity, as also in regard to vice, poverty and crime, and measures considered for their control come under the designation of preventive treatment. Steps taken to prohibit the sale of alcoholic liquor are prophylactic measures in regard to insanity, as are also those relating to the marriage of neurotic and insane persons, which in the opinion of many should be prohibited. The question of betrothals and their suspension is too long and complicated a problem to be discussed here, but this special aspect has recently been before the public in the effort made to establish the science of Eugenics or stirpi-culture—the propagation of the most fit—which is the direct and conscious application to man of the law of natural selection. Further preventive measures may be considered in the dissemination of knowledge as to the common causes of insanity and the means to be adopted for its avoidance, more especially by the treatment of incipient symptoms of mental disease in the out-patient department of our hospitals, where prompt and skilful advice would be given; also by assistance or employment given to those who have recovered from mental disease, as many of these not only fail to find work, but also suffer much hardship which not infrequently causes them to relapse.

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[1] A lecture delivered at the London Polyclinic, Feb. 14, 1905.

What should be done when a person not previously insane is threatened with symptoms of mental disorder? In many cases the treatment is obvious. The exciting cause, if any, must be removed, overwork must cease, overworry must be controlled and anxiety and overexcitement in regard to religious matters for example—to take a subject which at the present time is commanding public attention—should be stopped, undue devotion must be restrained, and life generally must be on a less exciting and stimulating plane. As to the remedial and therapeutic treatment of insanity—which is the theme of our lecture—we are confronted with the portentous fact that on January 1, 1904, there were 117,199 persons, i.e., 1 to every 288 of the population, who were certified or registered as insane in England and Wales; and speaking generally these were incarcerated under about 126,000 certificates (private patients necessitating two for their detention) amounting to more than five certificates at the hands of every registered medical practitioner, to whom therefore this subject must be of much interest and importance. We also know that possibly an equal number of persons are on the borderland of certification, and that out of this group fresh cases are being daily precipitated into those of whom the State—through the Lord Chancellor and the Lunacy Commissioners—has cognisance. In the acute stage of no disease is care and attention more necessary, nor more expensive, nor, may it also be said, is there more hope of cure than there is in the early stages of acute insanity. Unfortunately, however, the study of insanity is a long and painful experience, it is also as interesting in theory as it is trying and exacting in practice. The causes of insanity are, again, so complex, both so direct and so indirect and numerous, the "tissue of mind" is so complicated, its anatomy, physiology and chemistry are so little understood; furthermore, the very nature of mind itself is so incomprehensible and mysterious that our treatment of insanity not infrequently amounts—as is also the case with many other diseases—to the treatment of symptoms as they arise, i.e., to maintaining the strength of the patient by judicious nursing, to quieting hyperesthetic senses, to raising the hopes and spirits of the despondent, and to promoting their general comfort. Possibly in no disease is it more necessary to support the patient's strength by appropriate nourishment, for the abnormal rapidity of disintegration which occurs in acute insanity impairs nutrition so quickly that death from exhaustion often takes place before the delirium subsides. It becomes imperative therefore to obviate this most frequent cause of death by a vigorous administration of unirritating food—easy of digestion and nourishing—to be given frequently and in small quantities—"little and often." Foods, such as raw meat, or beef juice, peptonized beef tea, the yolk of eggs, milk with soda water or with biscuit powder, barley water, gruel, rice water, broths, arrowroot, cornflour, or Mellin's Food; all these being eminently suitable, possibly also, if delirium and exhaustion are profound, dry wines; also meat juices, but especially if the patient takes exercise out of doors. It is most necessary in all cases of acute insanity (acute delirious mania, acute puerperal mania, acute restless melancholia, etc.) to supply—even forcibly with the tube and funnel by the mouth or nose two or three times daily at fixed intervals—alimentary material to counteract the dangerous profound exhaustion, and also to renovate and maintain power, for unless sustaining treatment is secured, the exhaustion may prove fatal in a few hours. The judicious selection of food material is also important in regard to sleep. Loss of sleep is one of the most distressing and prevalent symptoms in the early stages of insanity. In more than one case of puerperal insomnia I have known an attack of insanity to be prevented by treating such secondary conditions as relieving pain, moderating uneasiness, allaying anxiety of mind, soothing irritability and inducing sleep, for absolute repose is necessary and must be obtained by the use of drugs. Whilst condemning the excessive use of calmatives and hypnotics as unjustifiable in the treatment of insanity, I must express my belief in sedatives. The motor excitement in certain forms of insanity—possibly from the production of a special toxin—is so fatiguing as well as exhausting, and the relief is so marked by the action of certain drugs, that in exceptional instances the morbid phenomena constituting insanity are cut short by the action of sedatives. Whilst discountenancing their use by persons who find in them a temporary alleviation to relieve sleeplessness, and who moreover, in many instances have established a habit which may become a disease; yet it is not improbable that many persons have been saved from attacks of actual insanity by early treatment of this distressing symptom.

It has been maintained that the acute insanities are often the result of intra- and extra-neuronic toxins which act injuriously upon the nerve centers, and that the sedative effect of drugs is antagonized by these. It certainly is a fact that long-continued administration of drugs, such as sulphonal and the bromides, do require increasing doses to produce a given effect, which tends to show that diseased or damaged tissue is not so readily acted upon by sedatives, and that a larger dose is required by the insane than the normal person. Furthermore (a state of dementia may also be brought about through the noxious effects of these drugs upon certain of the cortical neurones. We know that different parts of the nervous system are acted upon differently by various substances, e.g., conium, curare, strychnine, the bromides, etc., but our knowledge of the reason for the action of drugs is unknown, and our knowledge of this action is only empirical. We know that restoration or anabolism takes place mostly during rest and sleep, and that in health action and reaction are always equal and contrary. In insanity there is probably some alteration, congenital or acquired, in the rhythm of waste and repair in the higher centers of the nervous system, but these centers by the influence of sedatives, can be placed in a condition favorable to recuperation, and it is rather the combination of these sedatives with other remedies of a more general character which yields the best results; those for instance which favor nutrition by diminishing the irritability of the stomach, or which assist its functions, such as pancreatin, pepsin, hydrochloric acid or papain, and those which also assist in the elimination of poisons from the skin, lungs, kidneys, and the alimentary canal—i.e., those which act upon the emunctory organs, as well as the organs of assimilation.

The first consideration therefore in regard to the treatment of insanity is to ascertain if there be any abnormal condition of the bodily organs, removal of which will favor the disordered brain functions, and instances are not infrequent, and I refer to the adolescent forms of insanity in anemic girls—when the restoration of bodily organs to their normal functions is followed by mental improvement. The re-establishment of menstruation and the cure of anemia mean in these cases mental recovery, indicating the necessity for the combination of sedatives with general remedies which help to restore and build up the various systems of the somatic whole.

Before approaching the pharmaceutical armamentarium I may point out that conduct which is violent, destructive and filthy in habits, usually determines the question of restraint; but if such conduct occurs in puerperal women, and shortly after confinement, or with fevers, accidents, or the onset of inflammation of important internal organs, or if it be due to alcohol, or to some known toxin; if it occurs in young persons, or it is due to some sudden and possibly one great overwhelming emotional disturbance, in these cases the physician and the friends must be tolerant, and the person treated at home as long as possible, or for at least a period of six weeks. On the other hand, if the patient be acutely suicidal or homicidal, or if the case be one of general paralysis with violence, seclusion under a certificate is then not only desirable, but necessary and obligatory. In my opinion no supervision can equal the treatment given in our best private asylums and mental hospitals, which are especially constructed institutions where the comforts of a home are fully provided, where there are no wide departures from customary surroundings upon which sensitive persons are so dependent for their happiness, and where a skilful and trained staff exercise the closest vigilance and supervision. In these homes individual treatment is carried out, and the great benefit to be derived from this direct influence cannot be overestimated. Should there be, however, as there often is, much feeling against certification, then residence as a voluntary boarder in a mental hospital can be arranged, but should there be an insurmountable dread of all institutions on the part of both the patient and friends, then the best must be made of an improvised plan of private treatment, and the patient will have to be provided for in a private house, preferably in the country—for walking in the open air is necessary—and the rooms should be on the ground floor; all risk from stairs, windows, cutting instruments, strings or cords, should be fully safeguarded against, as also risks from fire and water. At least two competent mental nurses, one for day and the other for night duty, should be engaged, and they should make a daily written report of the pulse, temperature, food consumed, excretions, medicines taken, the hours of sleep, exercise out of doors, and all new symptoms. Relatives as nurses are less to be dreaded in cases of melancholia than in cases of mania, but usually in both conditions it is much better to engage competent nurses.

The treatment of mental disorder which comes within the experience of the general practitioner resolves itself most often into the treatment of cases of acute excitement or uncontrollable fury, but it must not be taken for granted that insanity with excitement invariably means a condition requiring antiphlogistic remedies. In cases in which there is delirium, when the pulse is full, bounding and firm, and there are symptoms of sthenic inflammation, venesection may prove beneficial. I have used it only for the venous engorgement of the status epilepticus, and then with good results. I have used the wine of tartarated antimony for furious excitement, with vascular and cerebral congestion; and when combined with morphia it is very effective. Aconite is a useful remedy in sthenic maniacal states, and the exacerbations of recurrent insanity with cerebral hyperemia. I have also used ice-bags to the head, and continuous immersion.

The difficulty in keeping maniacal patients in a warm bath at the body temperature or at 100 degrees Fahrenheit, has been so great that to avoid constant struggling I have abandoned the practice. The exact physiological effects and the therapeutic value of long immersion are not quite understood. It has been considered to be "derivative," i.e., the cutaneous vessels dilate and thus lessen the quantity of blood in the nervous system, but it has also been suggested by some that the effect of warm baths is to increase metabolism, by others that the pressure of the water on the skin gives a tone to the cutaneous and to the general circulation which was previously deficient. The use of the "wet pack" has been urged in cases of insanity with excitement, but there are dangers accompanying it, and I do not advocate it as a reliable remedy, although for certain bodily conditions accompanying insanity I have tried it with good effect. Insanity, even with excitement, is a disease generally of undernutrition, and whatever the pathological conditions may be which cause acute mania or acute melancholia, we can at present go no further than vaguely describe them as "a disturbed biochemical state" of the brain tissue, and my experience in the case of all the acute insanities recommends a generous and supporting rather than a depletory method of treatment.

In the treatment of incipient insanity by the medical practitioner the tendency to suicide must be regarded as ever present in cases of melancholia, and the question of "foreign travel" not infrequently comes up for consideration. Speaking physiologically the best brain work is done when the cortex receives abundant and definite stimuli from without. In travel, especially in the case of sea voyages, the life on board ship is exceedingly irksome, dull and monotonous, and should not be advised for cases in which there may be threatening of mental breakdown with symptoms of depression. In a voyage to Colombo, made by a distinguished writer, physician and man of affairs, he mentions two such cases, both of whom threw themselves overboard, whose lives would probably have been spared had they not been urged to travel abroad. Even trips to the Continent of

Europe do not provide an Englishman with the comforts he obtains at home, and which he hopes to receive abroad.

Electricity administered in the form of currents through water in the form of electric baths, has been greatly advocated for the depressed varieties of insanity, more especially for atonic stuporose states in young persons. The galvanic current is passed through warm water at 100 degrees F., in which the patient is immersed. I have used such for many weeks at a time, daily, or several times a week for about 10 to 30 minutes, and have kept a record of the weight whilst the patients were under treatment and found it to go up, and the general mental and bodily conditions to improve. I confess to being favorably disposed to this form of electric stimulation, and it certainly appears to aid metabolism—although how and in what method I am unable to explain.

As to the action of drugs upon the brain, it cannot be localized so accurately as can that of drugs acting on the spinal cord and nerves; but their action illustrates two important general laws. Firstly, the law of dissolution which shows that when a drug affects functions progressively those first affected are the highest in development, that is to say, they are the last acquired by the individual and the last to appear in the species. The next affected are those next to the highest and so on, until finally the lowest of all, from an evolutionary point of view, viz., the functions of respiration and circulation are affected. We are acquainted with this exemplification in the case of alcohol, for the first functions to be disordered are those of the intellect, especially the highest, such as judgment and reason; then follow disorders of movement and death from failure of respiration and circulation. Secondly, drugs in moderate doses excite a function, but in large doses they paralyze it. This is familiarly illustrated in cases of chloroform inhalation, the first effects being motor excitement followed by motor paralysis, the excited limbs becoming motionless and weak. In the same manner drugs which are cerebral stimulants may become hypnotics, as is exemplified in the use of opium. Conium also is one of the remedies which appears to have contradictory effects, but this is probably explained by the fact that the relation between conine and methyl conine—two alkaloids with opposite effects—varies in each preparation. It is an excellent remedy as succus conii (mx-ʒi), for controlling motor excitement. With the abolition of abnormal muscular action the ideas become less rapid and mobile, mental processes become clear and sleep occurs. It is best to begin with small doses and to combine these with strychnine or some other cardiac stimulant. As a nervous tonic for persons who suffer from overwork, or who are run down, who are weary and easily tired, the various preparations of phosphorus, more especially perhaps the glycero-phosphates, have in certain quarters had a reputation. On theoretical grounds, phosphorus should have been in much favor as a brain tonic, and it has been recommended for its efficacy in various forms of insanity, but the aphorism "without phosphorus—no thought" is not borne out by the experience of the clinical psychiatrist. The chemistry of the brain points out that lecithin, which enters largely into the structure of nervous tissue is a compound which breaks up into glycero-phosphoric acid and cholin or neurin, and in certain bodily states associated with convulsions it appears probable that the amount of cholin in the blood represents the amount of waste caused by the breaking up of nervous material in the discharge of energy. But the particular rôle of phosphorus in the brain has hitherto received no satisfactory explanation, and although the restorative nutrition of the brain is an important task for the psychiatrist, the problem of furnishing a reliable and assimilable preparation of phosphorus has yet to be solved; in consequence the various phosphates may be administered *ad nauseam* without modifying illusions, hallucinations, or delusions, and it is safe to assert in the words of Spitzka that phosphorus is not to the brain in insanity what iron is to the blood in anemia.

As to hypnotics, for the pure relief of insomnia, paraldehyde in doses of ʒss-ʒii (twice the amount is given for an enema) is one of the safest, for it stimulates cardiac action, and valvular heart disease is not a strong contra-indication. It has a peculiarly disagreeable taste and smell, which are best disguised in highly flavored syrups or wine. It is not recommended in phthisis or bronchial affections, as being volatile it is mostly eliminated by the lungs. Amylene hydrate, in doses from mx-xxx has been much recommended, and I have used it. Somnal, grs. 20-30, methylal ʒi-ʒiii, hypnone, chloralamide, chloralose, hypnal, tetronal, and trional, have also their advocates. I have used sulphonal grs. xx three times a day (insoluble in water), trional (less insoluble) and tetronal. These have a cumulative effect which is their best quality, and they are excellent remedies for strong, dangerous and aggressive maniacal patients. The effects of sulphonal do not appear fully for some hours, and as a night draught it should therefore be given early in the afternoon. The symptoms to be watched for are vertigo, ataxia, and hematoporphyrinuria. After death the blood appears to be fluid and to resemble dark port wine. The crystals may disturb digestion and cause diarrhea. If the patient is kept in bed during the administration, the physiological effects are intensified. Hyoscine in subcutaneous doses of 1-100 to 1-50 gr. is a powerful and valuable remedy for the control of acute motor excitement in violent maniacal cases, those who are strong and muscular and who in their fury may commit homicide. It differs from sulphonal and trional in that its effects are immediate, but they soon pass off. When the motor disturbances are controlled quiet slumber is produced. It is not a remedy for feeble cases. Hyoscyamine, dose gr. 1-60, combined with morphine is a very effective remedy for excitement and sleeplessness. Cannabis Indica is uncertain in its action, it gives rise to visual hallucinations and rapidity of ideas, the latter causing a feeling in the mind of indefinite stretches of time. When combined with bromide of potassium it is a useful remedy in senile insomnia, and especially when there is mental depression.

Possibly of all the remedies for insomnia the bromides are the most useful, for they lower reflex excitability and tend to slow down or diminish cortical activity. They are better hypnotics for states of excitement than for depression—but they may be combined with stimulants in such cases—and are therefore better suited for the insomnia of maniacal patients and for hallucinations of a tactile nature—paresthetic states as they are called—occurring in paretic and tabetic persons; also in cases of pharyngeal and laryngeal discomfort, giving rise to delusions of things in the throat. The effect of a single large dose is much better than that of long-continued under-dosing, and there is no danger of establishing a habit in regard to it. For alcoholic cases, for uterine reflex pains, such as occipital headaches, or in cases of neurasthenia with night terrors or unpleasant dreams, and combined with citrate of caffeine in cases of climacteric mental restlessness, the bromides are most useful. The only contra-indication is unsoundness of the circulatory organs and anemia, and the result of long-continued administration of bromides is a dull torpor with loss of memory and stupor.

Bromide of strontium is stated to be a better remedy for epilepsy than bromide of potassium and to be also safer aainst cardiac failure, which is apt to occur in epilepsy, and bromide of sodium is stated to be the least often accompanied by gastric irritability In association with chloral it is an excellent hypnotic for acute hallucinatory excitement, and possibly one of the best formulas is chloral hydrate, sodium bromide and tincture of hyoscyamus. The contra-indications to chloral are valvular or fatty heart disease, also if long administered with the bromides for epilepsy the tendency to cardiac failure from congestion arising in repeated fits should be remembered, also it loses effect by repetition. The dose of chloral is gr. x-xx, largely diluted, and flavored with aromatic syrups. It may also be given freely in milk. If given per rectum it is necessary to compress with the hand to retain the enema. The bromides are administered in doses of gr. xx-ʒi, or even up to one ounce and more. It is stated that neurotic medicines, such as are the bromides and also chloral, are best administered on an alkalinized stomach, hence a glass of milk is considered an effective adjuvant to such remedies. Possibly of all the hypnotics, opium, together with its derivative morphine, is the one most often used, and it is probably the best remedy for psychic as well as physical pains, such also as are due to painful "vagus" depression from cardiac crises and dyspnea. Opium relieves acute mental depression, especially in old people, and it appears to act as a direct stimulant in the cardiac weakness of some senile cases. It appears to act directly upon certain mental states, and to be antithetic to the painfully emotional states of melancholia and persecutory delusions. It is, in my opinion, contra-indicated in states of mania, but morphia may then be administered. Morphia is often the best anodyne for the precordial pain of melancholia, as also for painful hallucinations in exhausted cases. A combination of opium with belladonna may assist to prevent the impairment of digestion caused by opium taken alone, especially in regard to constipation. The great danger in the use of opium is the toleration established and the risk there is in commencing a habit which may become a terrible disease.

In contrast to hypnotics there are drugs which act upon the nervous system as sedatives, but stopping short of the actual hypnotic effects just described. Smaller doses of the hypnotic class effect this, but drugs such as valerian in large doses are known to control the agitation of neurasthenia or cases suffering from temporary functional disturbances, such as are seen and described as hysterical insanity. Camphor monobromate in g v doses has been used with benefit in cases of insanity with epilepsy, also in spasmodic excitement or mental disturbances accompanying chorea. Lupulin has also its advocates for cases of neurasthenia and hysteria, and the extract of calabar bean in general paralysis of the insane. The extractum physostigmatis was at one time much in favor, upon the recommendation of Fraser. I may be allowed to mention as a possible remedy for this disease the use of thorium hydroxide, a heavy white powder possessing radio-active properties. I have used 500 grammes of the powder in a cap over the head, worn day and night, and two cases have recovered after such treatment. It has been suggested that at the synapses—the arborization of the dentrites—there may be some radio-active body which either originates, transmits or intensifies nervous currents. If its possible modification in this disease is advantageously affected by thorium hydroxide the benefit may thus be explained. Possibly no direct effect is obtained, but at any rate it is worth a trial and something is being done for this hitherto incurable condition. In the congestive vertigo of this disease the fluid extract of ergot has been tried, upon the theory that it acts as a vascular stimulant upon the vasomotor center, causing a contraction of the cerebral arterioles. It has been recommended to be given every few hours during the day, and alternating with bromide at night. For the seizures occurring in epilepsy, general paralysis, and puerperal insanity, I have used chloroform inhalation and chloral, but prefer the latter.

I have used ergot for insanity about the climacteric period associated with menorrhagia. There are certain headaches and neuralgias about the "change of life" which almost incapacitate the sufferer from social and domestic duties. These so-called lancinating pains may be associated with mental restlessness and actual insanity, and no remedy seems to be of permanently good effect. I have used antipyrin as an analgesic in such cases in doses from g v to xx, but care is needed from the depressing cardiac effect, and antipyrin always needs a stimulant to follow. Phenacetin g x with quinia sulph. g i has proved a useful remedy, as also the citrate of caffeine in g iii doses. The peculiar restlessness of mental depression in regard to which there is no definite pathology, other than that it appears to have its basis in a summation of the organic sensations—and presumably caused by pneumogastric enervation—is often benefited by citrate of caffeine associated with small repeated doses of opium. It is in such cases that erythroxylon coca has been so much used, possibly also it is in similar cases that alcohol fulfils a definite therapeutic rôle, but it is necessary to beware of initiating a pernicious habit. As to alcohol, in the great majority of cases of insanity it is not essential, but I do not hesitate to use it in small doses every few hours in the exhaustion of acute mania, more especially in the puerperal form of insanity. It often interferes with digestion and it is always best administered with liquid nourishment, either with milk, soup, beef tea, or broth. In acute insanity it is often difficult to get the patient to take solid food, but by coaxing he may be persuaded to take fluids, and alcohol may thus be effectively combined with them. Alcohol increases the force and frequency of the heart's action and stimulates vascular tone, but in large doses alcohol acts as a definite depressant. Owing to this physiological action, it is a dangerous remedy in the various forms of mental depression, for it gives the feeling of buoyancy and stimulation without any lasting benefit and without the sustaining action of food. In the excitement of mania the skin acts more freely as an eliminating organ than it does in depressed mental states, and more alcohol can therefore be tolerated in cases of mania. Strychnia gr. 1-30 sub-cutaneously injected, and digitalis in m x doses every hour for three hours, and then stopping, are useful in cases of exhaustion from acute insanity, also in cases of cerebral anemia as a cardiac stimulant, especially when forced feeding is being adopted. Strychnia affects the whole nervous centers and not the spinal cord only. Nux vomica m v may be used with much good effect in the convalescent stages as a general tonic. The prolonged use of strychnia or nux vomica is ineffective, and they are better given alternately for a few weeks and with about one week's interval.

In the earlier part of my paper I have referred to eliminating medicine. The chief eliminating organs are the skin, the lungs, the kidneys and the bowels. In no morbid condition is it more important to clear the *prima via* than it is in insanity. Laxatives and purgatives are absolutely necessary, and in free doses—usually twice the amount required by a healthy person. Depressed mental states, irritability, and even the convulsive seizures in cases of general paralysis or epilepsy are often due to auto-intoxication caused by constipation, and consequent absorption of poisons from the alimentary canal. Constipation indeed is the bane of insane persons, and podophyllin, elaterium, colocynth, calomel, and at times even croton oil are necessary in full doses to cause a free action of the bowels. The hydragogue cathartics can be freely administered in cases of insanity, and "House Mixture" so called—the pharmacopœial Mist sennæ co. is a safe and salutary corrective in such cases and may be systematically administered once or twice a week with much benefit. The action of the skin and kidneys is assisted by the administration of diuretics and diaphoretics, but the best of medicines is exercise out of doors, which causes the lungs and skin to act freely and naturally and which facilitates the exudation of substances noxious to healthy mental action.

It is hardly necessary in such a lecture as this and before such an audience to emphasize the study of the various diatheses, i.e., to know our patient with his various idiosyncracies, viz., his tendency to gout, rheumatism, anemia, renal or cardiac disease, phthisis, myxedema, ague or syphilitic lesions which all require special treatment. The question of organotherapy and serum therapeutics is also too wide to be discussed within the compass of a lecture such as this, but I will summarize my suggestions by recommending in cases of sudden or acute insanity. Firstly, the isolation of the patient in a quiet, or in a darkened room; then forced alimentation; the control of motor excitement; reducing febrile states by tepid baths; sustaining the strength; and above all the inducing of sleep; these to be the cardinal lines of our treatment.

In conclusion, I may add that insanity may be modified in a community by the inspiration of high ideals, by agencies which are beneficial to sobriety and self-respect. I mean moral agencies. These kindle a spirit of fervor, sympathy and rightmindedness among the roughest characters, and in the most crowded areas of our cities. They are means which are peculiarly within the reach of medical men, who have opportunities of furthering them such as are afforded to no other profession.

Society Proceedings

THE BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

December 15, 1904.

The President, Dr. Blumer, in the Chair.

Wanted—A Reception-Observation Hospital in Boston.—Dr. E. Stanley Abbot read this paper.

Boston has no adequate place for the first reception of emergencies, such as delirium tremens cases, epileptics, persons becoming suddenly violent or dangerous through insanity, suspected insanity, or drunkenness, or for such other cases as require time and close observation to determine what kind of public institution is best suited to their needs. Boston's method of handling and places of detention for these emergencies were described. All such cases are taken to the City Prison, where they remain in clean, though unhygienic, surroundings under the care of police officers or matrons until they are discharged, committed to one of the State hospitals for the insane or for epileptics or for dipsomaniacs and inebriates, or sent for treatment or further observation to the hospital connected with the Penal Institution at Deer Island. The delay in thus distributing them may be two or three days, usually not more than one. Most of these cases need hospital care, such as is found in the acute wards of a hospital for the insane. The reader then briefly described the Bellevue Insane Pavilion in New York, which is a mere reception-distribution station, where patients nevertheless receive efficient hospital care during their two or three days' stay; the detention wards at Blockley, in Philadelphia, where patients are kept under hospital conditions for a day or two to three weeks; and the Pavilion F of the Albany Hospital, where patients are sometimes kept as long as six months; and referred to the "psychopathic hospitals" of Germany. The emergencies arising in those communities are well cared for, because hospital conditions, not prison conditions, are supplied. Three methods of providing adequately for the needs of this class of cases were outlined:

First, to provide a small hospital of twenty beds in the heart of the city for a mere reception-distribution station.

Second, to provide a larger hospital of thirty or forty beds, also centrally located, in which delirium tremens and other suitable cases might be kept for ten days to two weeks. This or the other hospital would be better under the direction of the Institutions Registration Department, which already has the care of these cases.

Third, to have provided by private beneficence a psychopathic hospital of one hundred to one hundred and twenty beds, near the new Medical School building and under the direction of the Harvard Medical School, where the clinical material might be used for teaching. In such a hospital not only could the needs of the dependents of this class be supplied for Boston and its environs, but also an urgent need in medical education could be satisfied. Hence the latter plan is the best, and the coöperation of the society, the school, the medical and benevolent public to secure the desired end by some one of these three methods was strongly urged.

Dr. Cowles said that the present conditions in Boston in the care of emergency cases of illness or injury are greatly improved since thirty years ago. The perfection of the provisions since then established for general hospital cases is now in strong contrast with the defective arrangements for mental cases. While our general hospitals and the custodial and medical care of the insane in our State hospitals have improved remarkably in this country, we are weak in two points: (1) the proper care of early and doubtful or emergency cases of mental disorder, and (2) the regard that ought to be given to research and instruction in psychiatry in our medical schools. In regard to teaching, the greatest progress has been made in Germany. There, through an adjustment of the relations of the local governments with the twenty universities, each of them is definitely provided with a special department in some form for giving clinical instruction in mental diseases in immediate connection with the medical department of the university.

The deficiencies in respect to the immediate care of emergency cases and those in the early and doubtful stages have been pointed out in Dr. Abbot's paper as they exist in Boston, and require first attention. There is besides this much to be said as to the kind of a hospital and its proper location that would meet other practical requirements of the larger class of cases and at the same time take full advantage of the present opportunity for giving the study and early treatment of insanity the proper place demanded by its great importance in connection with medical instruction.

It is reasonable to aim at doing the best that can be done. In respect to this larger purpose the practical question is: What now appears to be the best type of a special observation hospital? The working out of this problem in the German clinics, and a few beginnings in this country, have demonstrated very clearly the requirements, as shown by definite examples, of the ways in which provision for them has been practically made. They fall into four classes, in different degrees of desirable completeness.

(1) Small emergency or detention houses, or receiving and distributing stations for temporary care of persons taken in charge, or under protection by the police; such arrangements are essential in every city, and are the least that should be provided.

(2) Special wards or rooms in general hospitals; patients can stay in these places but a comparatively short time, especially those that are delirious and disturbing, though sometimes not really proving to be cases of insanity.

(3) Separate pavilion wards of general hospitals; these permit longer stay, but are deficient in means for classification, forbidding to certain classes of voluntary patients, and unsuitable for following the later developments of the early acute and curable conditions.

(4) Small independent special hospitals, standing somewhat apart, at least, from other hospitals in grounds with space enough to be made attractive and suitable for outdoor exercise, accommodating 60 to 120 patients in two or three small pavilions for each sex, having a central administrative house, with offices, lecture and examination rooms, and sufficient laboratory facilities for immediate clinical needs. Such a hospital with its surroundings, as simply built as practicable and equivalent in size to many of the small general hospitals now becoming so common, could be made sufficiently comfortable to attract voluntary patients, some of whom would contribute to the support of the hospital and gladly seek it for the observation and early treatment that might avert a graver illness in a hospital for the insane.

Most of the earlier German clinics, nearly all having been founded within the last thirty years, were begun in the forms of the first three classes above mentioned. Experience has guided the development of these special hospitals in two directions: (1) the enlargements of those which are adjuncts of general hospitals, or are closely related buildings, on the block plan where the cost of land is high in large cities; or (2) the perfection of the plan of the fourth class above mentioned. Excellent examples of these are the more recent ones at Wurzburg, Kiel and Giessen. In these places the hospital, being separated sufficiently from the other clinics to have a large garden or a few surrounding acres of land, is from this fact able to furnish valuable conditions for suitable construction and treatment not otherwise attainable.

The city of Boston is so completely lacking in the provisions for the uses here described that the opportunity is unique for some degree of improvement, or for supplying the whole need in the best way.

Dr. Owen Copp said that progress in this State in the care of the insane during the recent past leads logically to the establishment of special hospitals for the treatment of acute insanity, substantially as advocated by Dr. Abbot's very instructive paper. The principle that the indigent insane are wards of the State has been accepted by the inauguration of the State care act, an important preliminary to any adequate system of care. Primarily attention has been given to the extension of the colony plan, to provide for as many as possible on the simplest and cheapest basis, and thus to husband our resources for the better treatment of acute cases. Dr. Copp hoped to see in the near future a small special hospital for such in connection with one or more of our State hospitals for the insane, and eventually with all of them. Of course, such special hospital would attain a greater and more highly elaborated development in Boston than at the State hospitals.

The situation as regards the early care and treatment of the acutely insane in a large center like Boston is complicated, and probably will require some time and persistent endeavor in advancing, step by step, to a full achievement. The first essential is the determination of the different factors entering into the problem, the formulation of a general scheme which would meet, when completed, the full need of the public, and agreement as to the first step immediately to be taken for carrying it into operation, with concentrating of effort thereon for the present. Probably the provision of such special hospital is the first step, and certainly an important part of the general plan, but is not the final solution of the whole problem.

There are three main needs to consider:

(1) Temporary care of emergency cases.

The State Board of Insanity has received reports from physicians practicing in cities and large towns in the Commonwealth which show that 402 out of 1,132, or 35.5 per cent., of the persons whom they committed as insane in a single year were confined temporarily in lock-ups, police stations or city prisons. The length of such confinement may be considerable if a Sunday or holiday intervenes, or during the absence of a judge on his annual vacation without making adequate provision for the performance of so important a duty. Aside from the impropriety of association of the insane with criminals, whose rights may also be infringed by the disturbance resulting from the noise and violence of the insane, such places are not equipped to afford proper medical attendance and nursing for the mentally sick, and perhaps in a critical stage of illness.

These conditions call for an emergency station, under hospital auspices, accessible day and night, and able to furnish suitable ambulance service and facilities for examination and temporary care of such cases until they can be distributed to appropriate places for final treatment. As a matter of course, every mental case passing into the hands of the police should be taken thither and properly disposed after examination. The extent of such need probably would not justify an independent location or management, and although suitable safeguards against disturbance of other persons would be imperative, a combination with some other branch of a hospital would be desirable for supervision and necessary for economy.

(2) Provision for the treatment of persons on the border line or in the

early stages of acute insanity, under the voluntary relation, without the stigma of insanity.

This would seem to be one of the functions of a general hospital, like the Boston City or the Massachusetts General, in order to place the patient ill with mental disorder on the same footing as the patient sick with ordinary physical disease, so far as the mental invalid is willing or can be persuaded by tactful management to consent to such treatment. Under no circumstances should the general hospital be converted in part into an insane hospital, in the general acceptation of the term. Turbulence and violence of the insane should be eliminated in the interests of other classes of patients; forced detention should not be permitted longer than the brief period required to provide for the safety and welfare of the sufferer. Such restriction is fundamental, because forced restraint marks the line of division between the hospital for ordinary illness and that for the insane. The stigma of insanity will inevitably attach to any institution, however benign and enlightened its regime, to which legal commitment as insane is possible. Patients and their friends will be reluctant and usually will refuse consent to go there until the idea of insanity is well defined and acknowledged. In the end, it matters little as to the name of the institution; the fact of forced detention will become known and will determine its character in the mind of the public.

The need which would be met in this way is constantly coming to our attention, and is as imperative as any claim now recognized in the work of a general hospital. Special wards, or preferably a separate pavilion after the type of Pavilion F at the Albany Hospital, should be available in connection with at least one general hospital in the city of Boston. An alienist service should be created, both in the house and out-patient department. Any patient affected with mental disease who is competent to make application for admission and does so willingly should be received, and should remain only under the voluntary relation. Regulations should be made rejecting unsuitable cases.

(3) A special hospital for the acutely insane, whose prototype is the German psychiatric clinic.

Insane commitments should be allowed, although voluntary admissions should be encouraged and the spirit of voluntary relation should prevail so far as possible. The duration of treatment should be limited to a short and definite period, except in individual cases required as types for teaching psychiatry. Arrangements should be made for the easy and immediate transfer of chronic cases to the public asylums. A center for research and teaching on the highest plane should be developed, necessitating a location and association with the medical schools. The professor of psychiatry in one of them should be the medical superintendent.

Dr. L. Pierce Clark of New York, asked to make a few remarks upon psychopathic hospitals, said he thought there was no doubt that the ideal psychopathic provision consists of a distinctly separated hospital, complete and independent of any other hospital, but the practical way of acquiring that ideal would possibly be: first, through wards attached to some large city hospitals; second, through psychopathic pavilions adjacent to, but independent of, a large, centrally situated city hospital; and thirdly, and last of all, the real psychopathic hospital will be built. This is probably the practical light in which the taxpayer will look at the matter. It may also be the wisest and best course in the end for all concerned. Certainly many alterations will be necessary in evolving these hospitals in this country, although there are admirable German plans to guide us. The detached cottage construction, as at Kiel and Giessen, should be studied, most particularly the former.

Whatever provision is first undertaken, ample accommodation and special attention should be given to the borderland psychoses, epilepsies, hysterias and the like, especially from the treatment standpoint. These patients, as a class, are woefully neglected in many of the large dispensary and hospital clinics to-day. The doors of admission and discharge from the psychopathic wards and hospitals should swing easily; too much formality in either process will be very prejudicial to their success.

Finally, in agitating the formation of these provisions economy of the scheme should be pointed out to the taxpayer; to the patients, the advantages of treatment by this plan should be made plain, and to the public in general the opportunity of research and study into the best prophylaxis against the present appalling increase of insanity should be the keynote for their establishment. He had nothing further to offer as regards the details in construction, arrangements and location of these psychopathic wards and hospitals not already brought in his recent paper before the New York Psychiatric Society.

Mr. Montgomery, architect, from New York City, said the preceding speeches had treated this subject so thoroughly that there is little to discuss besides the arrangement, cost and style in constructing a psychopathic hospital for Boston. In the arrangement of a psychopathic hospital it is advisable to have all the buildings connected by conduits. The buildings should be two stories, with high basements, and accommodate about twenty-five patients each.

The administration building should be in the center, with the reception buildings, convalescent cottages and nurses' buildings, male and female, on either side.

In the center, at the rear, should be the kitchen, laundry, boiler and engine houses. The isolation cottages should be at either side to the rear, as far as possible from the other buildings as is consistent with economic management. A small mortuary and chapel should be placed conveniently on the lot, a house and stable for the superintendent, and two or three houses for employees complete the list of buildings required.

The interiors should be arranged with the idea of absolute separation of toilets and baths from bedrooms and wards, and of numerous, but small wards, containing from two to four beds each. The maximum of space should be used for wards, and the minimum for single rooms.

Lifts should be placed in each building for the transfer of patients. Permanent baths for the control of excitable and maniacal cases, and diet kitchens, should be provided, together with linen and clothes rooms, and in convalescent cottages there should also be a small dining-room. In brief, there should be accommodation for 150 patients and the proper number of nurses.

The same ideas can be carried out in a pavilion adjacent to a general hospital. The building, as before shown, to be of two stories with high basement, but containing twenty-five patients on each floor.

An open ward for about sixteen to eighteen beds, and from seven to nine single rooms, with necessary baths and toilet-rooms, also permanent bath, reception, waiting, examination and physicians' rooms and laboratory, complete the requirements of each floor.

The basement can contain the out-clinic department, with electrical treatment, Zarnsdorff-Swedish movement and hydrotherapeutic baths, connected by lift with upper stories.

Wards containing twelve patients, to be placed in existing hospitals, would follow the same general scheme of reception, examination and laboratory, and with four single rooms, together with a ward for eight beds.

The cost of allowing for every necessity without luxury would, in the hospital before-mentioned, amount to about $2,025 a patient. This is almost the same amount already expended at Kiel. Cost of ground not included. By substituting less expensive materials Mr. Montgomery succeeded in reducing the cost to $1,050. This would include all the buildings previously mentioned, together with heat, light and power.

The cost of a pavilion would be from $400 a patient upwards, and for the ward possibly $200, without addition of hydrotherapeutic apparatus. The style may be of any kind that is simple, dignified, homelike, and free from the asylum type.

Dr. Geo. F. Jelly believed Dr. Abbot had covered in his paper the whole ground of the need of special provision for the classes of patients we have been considering thoroughly and well; and Dr. Jelly agreed essentially with Dr. Abbot. He thought the subject of the paper was one of the most important matters which had been brought to the notice of the Society, and hoped it would not be allowed to drop. He suggested that a committee of the Society be appointed to further the matter.

Dr. Channing moved that a committee of five be appointed by the Chair to take the matter of a reception-observation hospital into consideration and report at some future meeting.

Carried, and the Chair appointed Dr. Copp, Dr. Abbot, Dr. Cowles, Dr. Channing and Dr. Baldwin.

Dr. Knapp had opposed the plan of an observation hospital in the neighborhood of the Courthouse, brought forward several years ago, as but little better than the present accommodations in the Tombs, and, without a resident physician, liable to grave abuses. The present conditions were, of course, unsatisfactory and unfit. A considerable number of mental cases, some already insane, others belonging to the borderland class, come under observation at the neurological out-patient department at the City Hospital as well as at the mental clinic of the dispensary, and, in spite of Dr. Clark's slur at neurological clinics, were often the object of considerable study. The question was broader, however, than simply as to the care of these cases. With the exception of two beds at the Massachusetts Hospital, there is no provision for the special care and treatment of patients with any disease of the nervous system at any of our general hospitals. The abolition of the service for nervous diseases at the City Hospital some years ago was an unfortunate and backward step.

There are many patients with mental disturbance but not actually insane who object to going to an insane hospital, or even to a "psychopathic" hospital, who can be cared for in a general hospital with a special nervous service, and are as worthy of treatment as the patient with typhoid, or even, with all deference to our surgically hypertrophied institutions, as the patient with appendicitis. If the old service for nervous diseases at the City Hospital had not been abolished many of this "residual" class might well be received there, and he would personally be very willing to care for them.

In regard to patients who are actually insane, he felt that there was altogether too much red tape in admitting them to insane hospitals. The State Board of Insanity should seek to simplify the necessary procedure, and do away with the old-fashioned formalities. Voluntary admissions, now possible under the law, should be encouraged, yet to-day many of the State hospitals are unwilling to receive voluntary patients. The example of the McLean Hospital should be more often followed in this respect.

Of late the "psychopathic" hospital was assuming the character of a psychiatric fad—a hospital for the scientific study of newly-committed cases, for modern treatment and pathological and clinical research and clinical teaching. What are our present hospitals for if not for this very thing? The trustees of the Boston Insane Hospital had built hospital wards, at an expense of over $200,000, especially for the hospital treatment and scientific study of acute insanity and as a reception hospital. When he was on the board he had suggested the establishment of an ambulance service if it should be called for.

The "psychopathic" hospital for the City of Boston was built and ready, but red tape and legal forms barred ready admission to its wards.

Nerve Suture and Anastomosis in the Treatment of Peripheral Palsies; Facial Palsy and Obstetrical Birth-Palsy (Duchenne-Erb).—This paper was read by Drs. L. Pierce Clark and Alfred Taylor of New York (by invitation).

The hopelessness of recovery in chronic facial palsy either following mastoiditis or Bell's palsy, and also in cases of chronic Erb's palsy in children, led the authors to undertake secondary surgical treatment for both.

The method of treatment of chronic facial palsy is as follows: The peripheral end of the divided facial nerve was implanted into the sheath of the neighboring hypoglossal nerve, the facio-spinal accessory method was discarded for the former, as the associated action of the tongue in using the facial muscles is not observable, and also because the functional and structural connection existing between face and tongue is closer than that of face and shoulder. The plan of lateral implantation results in the minimum damage to the patient and permits of movements in both face and tongue, although obviously the power of the hypoglossal nerve is divided between the two groups of muscles. The operation is begun by an incision along the front of the mastoid process and sterno-mastoid muscle; the parotid gland is pushed forward and the digastric muscle pulled downward. The facial nerve is divided in the stylo-mastoid foramen. The internal jugular is displaced forward and the hypoglossal secured. The lateral implantation is then done and the wound closed. (See *Medical Record*, Feb. 4, 1904.)

As for results: There was partial paralysis of the hypoglossal in all three cases, which lasted a number of weeks. After three or four months the face became symmetrical while at rest. In eight or nine months slight return of voluntary movement appeared. After thirteen months nearly full voluntary control of the formerly paralyzed side of the face returned. There was also slight evidence of emotional symmetry in two cases. The third case was lost to observation before the third stage of improvement could have occurred.

After clinical study of a number of cases of obstetrical birth-palsy, followed by manipulations and dissections upon twenty or more cadavers, and as a result also of detailed microscopical study upon the excised nerve fragments of the damaged parts of the brachial plexus in five of the seven cases operated upon, the authors conclude that the etiology of the chronic type of the palsy is invariably a process of excessive nerve stretching which occurs during birth. Its pathology is in accord with its etiology.

Seven cases of brachial birth palsy were operated upon, ranging from sixteen months to eleven years of age. The lesion was found to involve the fifth and sixth cervical nerves, as had been proven clinically. The best time for operation is about one year of age.

The incision is carried from the lower third of the sterno-mastoid muscle to the outer third of the clavicle, and is rapidly deepened to the plexus. The damaged tissue is mapped out by palpation, is excised and the nerves united by end-to-end suture. The wound is closed and the head and shoulder are kept in close apposition for three weeks.

As for results: Of seven cases two died; one within twenty hours, without infection and with many of the symptoms which are said to be due to lymphatic diathesis. The other case died on the third day, from recurrent diarrhea and anuria. The remaining five cases, ranging from sixteen months to eleven years of age, showed no reaction whatever, so that in a sufficiently large number of cases the mortality will probably be very slight.

Immediately after operation there was paralysis of all muscles supplied by the roots divided in the operation (fifth or fifth and sixth). After six months power began to return and improvement continues steadily. There is marked improvement in the nutrition and general condition of the limb in two cases. Sufficient time has not elapsed in the other cases to determine the final results.

CHICAGO NEUROLOGICAL SOCIETY.

January 5, 1905.

The President, Dr. Sydney Kuh, in the Chair.

A Peculiar Case of Lack of Orientation and Occipital Lobe Disease.—Dr. Patrick presented a man fifty-three years of age, a carpenter and ranchman by occupation. He had never been very strong, and he had had repeated attacks of what he called "lung fever"; that is, acute pulmonary attacks, probably not pneumonia. He denied all venereal disease, and had been fairly well able to work as a carpenter, farmer or ranchman until December, 1900, when he "took a violent cold." He had been working in wet and cold on a ranch in Nebraska, shoveling corn to feed hogs, and got his feet and legs wet. Suddenly he was taken with what he described as severe pain in the back of the head and back of the neck. He afterward said that this was rather a feeling of intense cold—so intense as to constitute a pain. He went into the house and tried various means to relieve this cold pain. After several hours, application of heat by means of bags of heated salt gave some relief, but for several days he had a cough, and for two or three weeks a terrific headache in the back of his head. Whether he was delirious or whether he had a very high fever he does not know, but he was confined to the house for about three weeks. The pain in the back of the head continued in less degree until about a year ago, since when he has had more or less dull frontal and temporal headache, but no severe pain.

When he recovered from this acute attack, which to him did not seem such a strange or peculiar illness, he noticed nothing in particular until he went out to the feed lot and about the stables. Then he experienced a very peculiar difficulty. The "feed lot" was an enclosure of about ten acres where were the stables, sheds and other outbuildings of the ranch, and a number of "feed bunks," or troughs, for feeding cattle and hogs. The house was separated from the feed lot bv a road.

After working in one part of the lot, on wishing to go to another he had no idea as to which direction he should take. This confusion was especially marked if he was working at the feed bunks, which were scattered about without order. He would look all around, searching the horizon for a landmark. Having located the house or the horse stable, he could approximate the proper direction, and thus, after a fashion, was able to go about his work. When out on the range, an open, undulating prairie, out of sight of the buildings, he was completely at a loss and could not find his way about. If the expression may be allowed, he was totally "at sea"; almost without orientation.

In consequence of this disability he lost his position, and since last August has been living in a Chicago suburb of about 1800 inhabitants. In this suburb he was born and reared, and lived there until he was twenty-eight years old. Yet, in this village he gets lost if he leaves the home of his brother, where he is now living. When taken to the house in which he had lived for many years, he recognizes the structure from certain features which he remembers, but has no conception of the relation of this house to the remainder of the town, and cannot go alone from it to his present residence, about half a mile away. Last fall, when he went a couple of hundred yards back of the house to dig potatoes he had to be conducted to and from the house.

When, in answer to leading questions, he tried to explain exactly the difficulty he had on the ranch and in the feed lot, he gave a very good description of a man with greatly restricted visual fields. He could see nothing except what was directly in the line of vision, and had to turn himself about and sweep the entire horizon and near-lying country for some landmark.

This restriction of the visual fields also causes difficulty in walking, because he is uncertain about the conformation of the ground. Although he can see it, he sees it indistinctly and cannot estimate accurately the height of steps or other irregularities unless he puts his head forward and looks directly at the place where he is to put his foot.

An examination was made roughly of the visual fields, and Dr. Mortimer Frank verified the results by a perimetric examination. Recognition of colors is restricted to central vision. For objects there is homonymous irregularly quadrantic vision. It might be described as homonymous hemianopia with the additional loss of the upper quadrant of the hemianopic field. Another peculiarity is that at the extreme periphery of the blind field there is some vision for five or ten degrees.

The great restriction of the visual fields explains in large measure the patient's difficulty in getting about, and at first it was supposed that this embraced the entire disability. A little further investigation showed that he has, in addition, impairment of the sense of orientation, whatever that may be, and impairment of visual memory. But this defect of visual memory pertains especially to localities, location and directions. The appearance of single objects he remembers fairly well, and even the arrangement of objects in a given locality he remembers to a certain extent. For instance, he drew a diagram of the feed lot on the Nebraska ranch, and located in it the various buildings, water tanks, etc., and from the way in which he did it, and in which he verified his own diagram a few weeks later, the plan seemed to be practically correct.

His greatest practical difficulty is in locating himself in reference to his immediate environment. Even in the house where he lives, which is neither a large nor a complicated residence, he is more or less confused. If, after reading for a time, he wishes to go to another part of the house, he cannot start off directly in the proper direction, but has to look all around the room, locate familiar objects, and then conclude which way to go. Making out the points of the compass is a laborious process.

In addition to this confusion dependent upon lack of orientation, he appears to have impairment of memory of things seen. Even allowing for the fact that he sees many things indistinctly, and that it is easier to see them indistinctly than to make the extra effort to get them all in turn into the fixation point, he does not remember sufficiently well objects which he does see clearly.

For instance, he was sent into an adjoining room with instructions to note carefully everything that was in it and then come back and enumerate them. He took particular pains to note carefully every object in the room, but was unable to enumerate them ten seconds later. Of seven objects noted he forgot three. Fifteen minutes later he had a still more imperfect recollection of them. Apparently he does not remember faces as well as he should, and is entirely unable to recall houses, rooms, stairways and similar things with which he should be more or less familia. He says that he does not remember well what he reads. He remembers much better what he hears, but says that in all respects his memory is not as good as it was.

The examination reveals remarkably little in addition to the peculiar visual fields. Central vision is good except for an error of refraction which he has always had. The fundi are normal, the pupils are equal and react perfectly to light. Hemiopic reaction has not been examined. Physical examination reveals nothing, except that the wrist-jerk is a little more brisk on one side than the other. Motion, sensation, coördination, thoracic and abdominal viscera are normal. There is no arteriosclerosis. Aside from vision the special senses are unaffected.

He did not state that he could not see objects on one side or in certain directions, but when questioned about it said that when moving objects approached him from one side he did not see them until they got to the middle. In his own statement he did not complain particularly of his failure of vision, but that he got lost and confused, could not find his way about, and consequently, could not make a living.

It seems clear that he has a bilateral occipital lesion such as to cause disappearance of the visual field on one side and disappearance of practically the upper half of the field on the other side. Lesion of any other part of the visual tract seems to be out of the question. Dr. Patrick thought the visual center on one side was destroyed, and that he had a small lesion on the opposite side, undoubtedly on the mesial surface. According to Beevor and Collier's[1] recent article, this would be of the lower part of the cuneus and probably of the lingual lobule.

Dr. Patrick was at a loss to determine what the lesion was. It seemed quite unlikely that it was caused from vascular occlusion, as the conditions which would produce thrombosis or embolism seem to be absent. It was probable that the acute illness which he had when this trouble appeared was an infection of some sort, and that the infection produced the localized encephalitis or meningitis. This might be due to either the germ of influenza or the pneumococcus.

As regards treatment, no measures directed against the lesion itself will be of any avail, but some educational procedure might be of benefit and this would be tried.

Dr. Sanger Brown said he would like to ask if since this trouble the patient had found that hardly anything seemed as it did before. Had he been able to enjoy that sense of knowing where he was and appreciating things just the same as before?

Dr. Patrick said he had not. He felt very much "at sea" all the time. He got confused even in the house. Things that should look familiar seemed unfamiliar. And this can scarcely be explained by the defect of vision alone.

Dr. Mettler asked what was the objective sense of orientation; when he shut his eyes could he see things as they ought to be?

Dr. Patrick replied that he had asked him what was in the sitting-room at home. He mentioned most of the objects and their location, but forgot the couch. There were not many things in the room. The patient said that when he came back to where he lived as a boy he could not see a thing, except one, that seemed familiar. He had been away twenty-three years, but could recognize only an old tree where he used to have a swing.

Dr. Brown asked the patient whether, when he had dreamed since his sickness, he had dreamed of things in their natural relations.

The patient replied that he had not dreamed in twenty-five years.

Dr. Brown asked him whether he could remember scenes in his early life, how objects looked, recall them as he did before he was sick.

The patient replied that he could.

He was asked whether he could remember who wore glasses, for instance, and the peculiarities of his playmates?

He replied that he could.

Dr. Sanger Brown said: In assuming that there would be one lesion which had disturbed the visual power in the right occipital lobe; to assume that that lesion, whatever it was, passed over a little into the other lobe and injured the contiguous parts of the other lobe, would not very well account for what is found in his visual field, because there would have to be, in that case, a disturbance of central vision.

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[1] "A Contribution to the Study of the Cortical Localiation of Vision." Brain, 1904, p. 153.

Dr. Patrick said he thought not. Central vision is the last to go.

Dr. Brown remarked that there is some central vision in each cuneus. There is a double supply from the center. That is the pit or core of the whole thing.

Dr. Barker said the lesion must involve the entire occipital region, according to von Monakow.

Dr. Patrick thought a partial injury of the cuneus on one side would account for the quadrantic loss in addition to the hemianopia from a more extensive lesion of the other side.

Dr. Barker said the lesion must be in the lower part of the occipital lobe.

Dr. Patrick remarked that Beevor and Collier's conclusions do not entirely agree with other writers' as to the location in the lower part of the lobe.

Dr. Brown did not believe that it has been fairly proven that the cuneus has all to do with vision. He believes the visual area extends over the entire occipital lobe, but the ophthalmologists and pathologists who examine cases post-mortem seem to be of the opinion that the cuneus is the only part. He believed that the reason that the lesion in the cuneus is so effective in obliterating central vision is because it involves the fibers that go to the other parts. That is comparable, in some particulars, to internal capsule lesion in motor diseases.

Dr. Mettler said the case interested him from another side, the psychic side. There is a loss of space sense, and we are taught that we acquire that through vision. He had lost the fundamental space sense, which enters into the psychic section through his loss of vision, this orientation being a psychic loss—something gone out of the man's mind.

Dr. Kuh said that Berlin had published a case like this.

Dr. Patrick remembered that there was no indication of vascular disease; the heart was good, and the arteries showed nothing in the examination which would lead one to suspect the man had a thrombosis. He recalled three cases of occipital lobe thrombosis, but none like this. The man had been peculiarly susceptible to pneumonia.

Dr. Barker thought there was a spotty encephalitis, with marked destruction of brain substance. Most of these cases terminate fatally, but some do not. In this condition the influenza bacilli are present and both hemispheres are involved, but one end of the brain more than the other.

Dr. Patrick thought that would explain it better than meningitis.

A Case for Diagnosis.—Presented by Dr. D'Orsay Hecht at the request of Dr. H. T. Patrick. The patient was a man 36 years of age, who first came under the speaker's observation one week ago in Dr. Patrick's neurologic dispensary service at the Northwestern University Medical School. He was a carpenter by trade. His present disability had caused him to drop that occupation and for the past two or three years he had peddled, principally tea and coffee, for a living.

He had been married twelve years and was the father of a healthy boy. His wife had no miscarriages. The family history, so far as he knew, had been negative. His father was living and well at 62, and his mother enjoying good health at 58. Tne patient was the oldest of thirteen children, of whom eight were living; of these a brother and sister were said to have "enlargements of the neck" (perhaps goiter). As to the venereal history, both gonorrheal and syphilitic infection were strenuously denied, nor was there found upon examination any somatic evidence of past lues.

The patient had been a heavy drinker ever since his fifteenth year, indulging in both liquor and beer. Nine years ago he sustained a fall from a 22-foot-high scaffolding, attended with immediate loss of consciousness. Having been taken home in a patrol wagon and put to bed, consciousness was regained in an hour or so, and it is stated that he then passed considerable blood per rectum (according to patient a half-bucketful). Except for the occurrence of a few vomiting spells, the six weeks' term in bed following this accident revealed neither marked subjective nor objective symptoms. There was no paralysis of the extremities; no notable incoördination; no vesical or rectal disturbance.

His first attempt to get up and resume work was attended with great weakness in the legs and some dizziness. Vision in any direction engendered a feeling of marked and constant vertigo, which was not further aggravated by sudden changes of position of body or head. Sudden rising up or lying down did not affect it. Simultaneously subjective sensations of dark spots floating before the eyes were experienced. He had been singularly free from headaches. Transitory diplopia had been present. A return to his former occupation in this condition had proved a physical impossibility, and the patient became engaged in a bottle beer business, in the pursuit of which he had managed personally to consume as much as two cases of beer a day.

During the two years he was so occupied he was better or worse. In a state of drunkenness and while delivering beer he had met with another fall to the ground from the wagon seat, and the jar so received seemed to have increased his weakness, because his gait now became markedly unsteady and a speech defect (dysarthria) appeared for the first time. The bottle beer business was abandoned for an active interest in a pool and billiard room. His incoördination at this time must have still been fairly good, since he was able to make runs of three and six in three-ball billiards, and occasionally put down a half-frame in pool. He concedes that there might have been an element of good luck in his play.

For the past two years, which have been devoted to tea peddling, all symptoms, and especially those of incoördination, have been alternatingly better or worse. When away from localities in which he was well known he had been arrested several times for drunkenness, the impression readily justified by his extremely ataxic gait.

Although inclined to an occasional display of emotionalism, there had always been a decided sense of well-being, akin to euphoria—this despite acknowledged domestic infelicities and economic distress. To his own immediate infirmities he referred in terms of "feeling fine, never better in my life," etc. At no time had he presented spasmodic weeping or laughter. There had been no signs of mental enfeeblement and his memory remained unimpaired.

Physical examination revealed the following: Nystagmus-like twitchings of both eyes when in extreme lateral position (in no sense genuine nystagmus). The right pupil was somewhat larger than the left, both regular in outline and reacting well to light and accommodation, but none too promptly.

The visual fields were normal for distance and color. It had been Dr. Gradle's first opinion that the temporal halves of both disks showed a slight shade of pallor, but on second examination he had concluded the fundi were normal. The tongue was tremulous on protrusion. Dysarthria was marked—an admixture of syllable stumbling and scanning speech.

Of the motor symptoms the most conspicuous was the gait, decidedly cerebellar in type, broad-based, and purely ataxic, with lateropulsion to the left. None of the extremities were in the least spastic.

The marked incoördination was further demonstrated with patient placed upon his back and his legs raised in the air. Execution of this movement was attended with widely excursive, most disorderly swaying of the legs. There was absolute inability to maintain them in static equilibrium.

Approximation of the index fingers to the nose with eyes shut was impossible even after repeated trials.

Sensory phenomena, solely of the paresthetic variety, were confined entirely to the distal parts of both upper and lower extremities.

The patellar reflexes were only satisfactorily elicited with Jendrassik reinforcement—the right being somewhat weaker than the left. The Achilles jerks, also requiring Jendrassik's reinforcement, were present and more equal. The Babinski toe sign was absent. Of the skin reflexes, the plantar, cremasteric and abdominal were active on both sides. The anal reflex was present.

Referring to the diagnostic possibilities in the case, Dr. Hecht stated that a cursory examination of the most conspicuous features had suggested a diagnosis of pseudo-tabes alcoholica, which was abandoned immediately upon more careful consideration of the findings.

The diagnosis of multiple sclerosis had then seemed quite tenable, although there was nothing typic of this disease in either the eyes, the speech, the gait (total absence of spasticity), the reflexes (either deep or superficial), or the mode of onset. Some findings which Dr. Edward Müller in his recently published monograph on multiple sclerosis had emphasized to a degree of diagnostic import were totally lacking in the present case.

The possibility of cerebellar disease had received only passing consideration, because of the failure to reconcile the symptoms to a lesion in this location. In this connection, the speaker called attention to the conclusions drawn by Babinski from his studies of static and kinetic equilibrium in cases of cerebellar asynergy.

The Babinski "dissociation of the two kinds of volitional equilibrium," the kinetic and static, had not been demonstrated in this case, nor had the patient encountered any difficulty in executing rapid supination and pronation of the hands (diadocokinesia), a function supposed by Babinski to have its anatomic seat in the cerebellum. From all these negative premises, Dr. Hecht felt constrained to call this an obscure case, and invited the members to suggest a diagnosis.

Dr. Grinker, who had been privileged on two occasions to see the case, thought that Dr. Hecht had covered all the points brought out at a joint examination. He, too, had favored a diagnosis of multiple sclerosis, and upon first examining the disks thought the temporal halves had appeared slightly paler than normal, but a second look had disabused him of this belief. To eliminate all doubt, he had asked Dr. Gradle to examine the eyes, and he reported the nerve as slightlv grayish-white, but not pathological. The history of chronic alcoholism, the apparent reduction of the tendon reflexes, together with the gait, were highly suggestive of pseudo-tabes, but the absence of sensory symptoms weighed heavily against this conclusion. He said that he, too, was familiar with the Müller monograph on multiple sclerosis, which he had probed for case reports with reduced or abolished knee reflexes. The testimony on this point had not been convincing, nor had he ever seen a case in which the patellar reflexes were weakened or absent. He thought that this might be one of the very typical cases of multiple sclerosis. Müller had declared as essential to the diagnosis of this disease symptoms beginning early in life, with a slow and insidious onset. In this case, they appeared in adult life acutely and rapidly progressive following trauma. With such a history the diagnosis of a myelitic process (sclerotic areas) seemed not altogether unlikely, but he preferred to ask rather than answer the question, What is it?

Dr. Sanger Brown asked whether the ataxia had been as marked two or three months after the accident as it is now.

Dr. Hecht replied that it had been sufficiently great to prevent return to his trade, but had not so early advanced to its present degree.. For the seven years after the accident he felt decided improvement from time to time. Dr. Brown then inquired as to the patient's muscular strength and power. Dr. Hecht was able to demonstrate that the patient was in all respects strong, but that he tired easily, and frequently required a half hour's rest to put him right again.

Dr. Llewellys Barker asked Dr. Brown whether the speech defect in this case bore any resemblance to the dysarthria in the cases of cerebellar ataxia, described by them (Barker and Brown). Dr. Hecht had the patient recite the Lord's Prayer, commenting upon the rhythm, the tonal monotony, the confluence of some and the omission of other syllables. Dr. Brown then replied that the speech differed considerably, it having in their cases been more confluent, more guttural and grunting in character.

Dr. Sydney Kuh said that one possibility always to be thought of in an obscure organic disorder was that of specific disease. Although infection had been denied, the sort of life the patient led would not make the fact at all improbable. If it were his case, he would give the patient the benefit of the doubt; it could do no harm, and might clear up the diagnosis. At the Post-Graduate Hospital he had seen a colored man in a similar condition derive enough good from antisyphilitic treatment to enable him to return to his work. To him the diagnosis in the present case rested between syphilis and an atypical disseminated sclerosis, perhaps a pseudo-sclerosis.

Dr. L. Harrison Mettler looked upon the case as one of the atypical forms of multiple sclerosis, with alcohol as an underlying cause producing encephelo-myelitic foci simultaneously with the changes in the cord.

Dr. Barker felt that on account of the bizarre character of the case Dr. Kuh's suggestion to try specific remedies should be carried out. He did not care to venture anything approaching a definite diagnosis.

Dr. Grinker said he had deterred from making a diagnosis of specific disease because of the entire absence of somatic findings and the knowledge of a healthy family.

Dr. Hecht, relative to Dr. Kuh's suggestion of possible lues, put particular stress upon the total absence of headaches at any time in the history of the case, adding that the only headache the patient had ever experienced was (to use his own words) "the head of a jag."

Dr. Sanger Brown then referred to his publication three years ago of three cases of acute ataxia, in which the progressive asynergy acute in onset had been very much more pronounced than in this instance, and spasticity was recorded in all. He was able to observe them for a number of years. In one of the cases (that of an old lady) in which he had determined a cerebral lesion involving the red nucleus, there were symptoms of third nerve paralysis associated with those of hemiplegia. Some fibers of the pyramidal tract were involved in their passage through the pons. Another instance recalled was that of a man 23 or 24 years old, still living, who had not walked for years, whose strength had not diminished a particle, whose speech was more affected than this man's, and whose incoördination was greater.

In the case now under discussion, he ventured the opinion of some lesion not in the cord, but in the cerebellum, which had thrown it (the cerebellum) out of circuit. He thought that in view of the history of trauma and the acute onset of symptoms in an otherwise healthy man, the diagnosis of specific disease was far-fetched.

Periscope

BRAIN

(Vol. 27, 1904, No. 106, Summer.)

1. A Contribution to the Study of the Cortical Localization of Vision. A Case of Quadrantic Hemianopia with Pathological Examination. C. E. Beevor and James Collier.
2. Some Cases of Family Disseminated Sclerosis. Ernest S. Reynolds.
3. Fecal Vomiting and Reversed Peristalsis in Functional Nervous (Cerebral) Disease. A Summary of Cases and Conclusions. F. Parkes Weber.
4. Intramedullary Abscess of the Spinal Cord. An Account of Three Cases. William Alden Turner and James Collier.
5. Bilateral Loss of Postcentral Cortex, Apparently Congenital, in Adult. Hubert M. Turnbull.

1. *Cortical Localization of Vision.*—The authors have investigated a case of quadrantic hemianopia and present the results of a pathological examination with microscopical studies. The patient during the greater part of the time that he was under observation showed a partial blidness in the left upper quadrant in both visual fields, the fixation point escaping. This condition had remainsd constant for a period of nearly two years. An occlusive lesion of the right posterior calcarine artery had caused destruction of the cortex: (1) of right fusiform lobe posterior 2 cm.; (2) right lingual lobe from junction of calcarine and parietal occipital fissures to the pole of the hemisphere; (3) of the whole cortex in the depth of the calcarine fissure; (4) of the greater part of the inferior cuneal gyrus, small areas only at the anterior and posterior limits of the gyrus being free. The necrosis did not involve the optic radiation at any point. The only parts of the cortex of the occipital lobe which had escaped destruction were the upper two-thirds of the cuneus and the anterior and ventral portion of the fusiform lobe. The lower quadrants of the visual fields were entirely unaffected, and until it was pointed out to him the patient was unaware of the visual defect. The authors submit, therefore, that this case affords conclusive evidence that the cortex of the upper two-thirds of the cuneus is the primary visual center for the lower quadrants. The following inferences are drawn from the case: (1) The larger part of the lesion was situated below the calcarine fissure. The upper quadrant is chiefly, if not entirely, represented below the calcarine fissure. (2) The cortex lining the calcarine fissue was completely necrotic. The primary half vision center cannot, therefore, be limited to the calcarine cortex nor the macula exclusively represented in the anterior part of this region. (3) The portion of the half-vision center, as limited by von Monakow, which was not involved was the upper two-thirds of the cuneus. (4) The visual defect was blindness of both left and upper quadrants. The lower quadrants are represented to a great extent in the upper two-thirds of the cuneus. (5) The lower quadrants of the visual fields were not affected, and the patient was not himself aware of any visual defect.

2. *Family Disseminated Sclerosis.*—The authors put on record two instances in which this condition may be said to have affected more than one member of a family. In one family the father is suffering from melancholia, the first cousin of the father died of disseminated sclerosis. A sister of the mother suffered from melancholia. There were five children. The

eldest, female, died from melancholia; the second, female, had multiple sclerosis; the third has severe sciatica; the fourth is one of the cases reported; the fifth is well, and the sixth is the third case of multiple sclerosis reported. The second family consists of three daughters, M., C. and J. M. The eldest, 29 years old, suffers from advanced multiple sclerosis; C. is in good health, and J. M. is reported in the history as another case of multiple sclerosis. These patients are all suffering from irregular types of the disease.

3. *Reversed Peristalsis in Nervous Diseases.*—F. P. Weber presents an interesting summary of this condition, from which he draws the following conclusions: (1) Functional nervous vomiting, like the hemianesthesia, palsies and spasms of hysteria, must be regarded as due to an abnormal state of the cerebral cortex, and is just as much a symptom of functional brain disease as the vomiting in cases of cerebral tumor is of organic brain disease. (2) Fecal vomiting of functional nervous origin is merely a rare and extremely exaggerated form of ordinary hysterical vomiting. (3) The vomiting in functional brain disease may sometimes be more violent and severe than it ever is in organic cerebral disease, since fecal vomiting is scarcely known to occur in cases of cerebral tumor, etc. Some light is thrown on this point by the fact that a delusion is apt to be more stable and better "organized" in a monomaniac whose brain, could it be examined, would probably show no obvious change, than in a general paralytic whose brain is the seat of grave organic disease. (4) For the occurrence of fecal vomiting of functional nervous origin active intestinal antiperistalsis is absolutely necessary. But it is not certain that antiperistalsis necessarily always plays a part in the fecal vomiting known to surgeons as a symptom of organic intestinal obstruction (organic ileus). (5) The fecal vomit in organic obstruction of the bowel is seldom, if ever, more than "feculent"; that is to say, having the odor of feces without containing visible fecal particles or masses. Vomiting of formed feces in the absence of malingering and gastro-colic fistula practically only occurs in functional nervous cases. This may partly be accounted for by remembering that antiperistalsis, if it occurs at all, is likely to be more forcible when the muscular walls of the gut have not been previously weakened by overdistension or gross organic disease. (6) Hysterical malingering is, of course, apt to develop in the same (hysterical) class of patients in whom fecal vomiting occurs, and the possibility of genuine fecal vomiting occurring side by side with simulation must be kept in mind.

4. *Intramedullary Abscess of the Spinal Cord.*—Drs. Turner and Collier report on three cases of this rare condition following suppurative leptomeningitis which have come under their observation. The cases resembled each other closely, being cases of pressure paraplegia in which a transverse portion of the spinal cord was the seat of myelitis or necrosis from loss of blood. In all cases the suppuration commencing in the necrotic area spread the length of the spinal cord above and below. In the first case the paraplegia was slow in onset, and had been complete for several weeks when suppuration occurred. Though it is certain from a pathological examination that the suppuration occurred subsequent to the evascularization, nothing in the clinical history of the case seemed to indicate at what period suppuration commenced. The physical condition was feeble. Rigors did not occur, nor was the temperature above normal throughout. There was no alteration in the spinal symptoms indicative of the upward extension of the abscess. In the second case the paraplegia became complete in a few days after the first appearance of spinal symptoms, and this was evidently the result of extensive strangling of the spinal arteries by pachymeningitis in the lower half of the dorsal region. The patient lived a month after the onset of paraplegia. The advent of suppuration was presumably marked by the occurrence of pyrexia and rigors, which persisted until death, but by no other change in the clinical picture referable to the nervous sys-

tem. In the third case the onset of paraplegia was rapid. The presence of pyrexia during the time the patient was under observation, and the fact that the spinal symptoms progressed as if from a spreading lesion, made it probable that suppuration rapidly followed the occurrence of myelitis and that the spread of symptoms was evidence of its extension. That suppuration commenced subsequently to the occurrence of the myelitis is, we think, clearly proved by the fact that in the limited transverse area of the compression myelitis the spinal cord was completely necrotic, and at this point the abscess was least in evidence, tissue reaction being possible in the necrotic area; but above and below the lesion, where the abscess was conspicuous and extended for many inches in the length of the cord, there was no sign of such necrosis. The paper is profusely illustrated.

5. *Bilateral Loss of Postcentral Cortex.*—An extensive description of a bilateral loss of the postcentral cortex, apparently congenital, in an adult 24 years of age, who died from severe burns. Life history was obtained from the patient's mother and sister. Patient was delivered in an easy labor. As a baby she had several infantile convulsions, and two severe fits with unconsciousness, one as late as her seventh year. She frequently had fits of temper and would scream and strike at people. These attacks never had any of the earmarks of the psychic equivalents of epilepsy. She never had any attacks of paralysis or transitory weakness. She was late as a child in her speech, and was able to converse rationally, but she was distinctly undeveloped. She was practically blind and imbecile from birth. Her hearing was acute, and she understood the significance of words, particularly of the sounds connected with the preparation of food. Her sense of smell and taste was distinct. She could smile when pleased, but never laughed heartily. She had control of her sphincters. She could never dress herself, do her hair or blow her nose, and had been growing distinctly weaker. Examination showed complete loss of the postcentral cortex on both sides, which space was occupied by enormous cysts, each cyst being covered by arachnoid and representing attenuated portions of the cortex. On the left the cyst was an evolution from the thin cortex of the limbic lobe immediately below the notch representing the anterior limb of the calcarine fissue. Thus the thin cyst replaced all the parietal lobe save the ascending parietal and one-half of the supramarginal convolution, practically all the temporal lobe save the pole and the anterior two-thirds of the supra-temporal convolution and the temporal operculum, all the precuneus on the left and posterior two-thirds on the right, the whole occipital lobe, including the cuneus and all but a small fragment of the lingual lobule. The abnormalities found by microscopic examination consisted of a distension of the ventricles and destruction of a large area of the cortex accompanied by certain changes in the remaining cortex, in the corpus callosum, in the retro- and sub-lenticular portions of the capsule, and in the optic nerve and its central connections. The facts settled beyond dispute in the study of the interdependence of parts of the central nervous system justify the statement that the changes in the corpus callosum, the capsule and the optic nerve connections were undoubtedly secondary effects of the cortical destruction, and suggest a similar explanation of the abnormalities in the thick cortex. The physical deductions were of considerable interest, and well worth quoting in toto.: "Finally, from the life history of the patient it is possible to estimate the physiological effects of the cortical destruction, and to see how far these agree with the present views on cortical localization. *The olfactory sense* was present and played an unusually important part in the choice of food, replacing the lost sense of vision. That it was not interfered with by the lesion was to be expected, seeing that all those parts of the brain were intact which, from the study of comparative anatomy and such experimental and clinical evidence as have been obtained, have been associated with the sense of smell. Those parts are: the olfactory bulb and tract, the uncus, the hippocampal convolution,

hippocampus major, dentate convolution, nerves of Lancisi, peduncles of corpus callosum, fornix, fimbria, anterior commissure and the callosal convolution. *The sense of taste,* the cerebral center of which is probably in close association with the olfactory, was also present. *The neuromuscular mechanism.*—The limbs could be moved at will, and the sphincters were under voluntary control. There was, however, latterly weakness in muscular movements and inability to walk without support. That the motor area of the cortex—applying the term to the area containing the cells which give origin to the pyramidal projection fibers—was not interfered with was shown by the perfect condition of the genu and the posterior limb of the internal capsule and of the pyramidal tracts. In this connection it is interesting to recall the condition of the ascending parietal convolutions. Not only were these absolutely and relatively extremely small, but on microscopic examination it was seen that, where the cyst had origin from this convolution, the adjacent portion of the convolution was entirely devoid of nerve cells. That such a condition of the parietal convolutions was compatible with an anatomatically perfect pyramidal tract is consistent with the observations of Sherrington and Grünbaum that in the anthropoid apes the postcentral convolutions are electrically inexcitable, and their injury is not followed by degeneration in the pyramidal tracts. No marked wasting was seen in the limbs and the anterior horn cells of the cervical and lumbar enlargements of the cord were healthy. It is reasonable, therefore, to suppose that the talipes equinovarus noticed in the necropsy was the result of long sitting in the tailor position and not of a spastic paralysis. It is difficult to understand why the patient, who had been able to walk spontaneously and unaided, lost that faculty; had the loss been accompanied or preceded by a fit or by a loss of muscular power it would have clearly pointed to a progression of the destructive lesion, but no loss of power could be noticed at the time and the behavior of the patient was such as to make it appear that there was a disinclination rather than an inability to walk. Her mother made no effort to overcome the disinclination, and it is possible that the power of spontaneous and unaided progression was ultimately lost through disuse. *Of muscular and tactile sense* little can be said. That she could lift and play with small objects, although blind, and could modify the force of her muscular efforts, for instance, using her hands to put food in her mouth, or striking violently when angry, pointed to the integrity of these senses in the arms at least. The sense of touch, however, did not seem to play an important part in her appreciation of the outside world. *As regards speech,* the neuromuscular mechanism of articulation was normal. The phonogenetic center must have been present, and this must have been in connection with the auditory center to enable her to speak. Not only did she recognize the sounds of certain words, but she associated certain facts with them, and could even associate sounds heard with words; thus, she would repeat "Annie is going to have some fish" on hearing fish being fried, so that she may be said to have possessed an auditory word center. The connection of the auditory word center and phonogenetic center, with the higher centers of intelligence and memory, seems to have been present, in that she could answer certain questions correctly in the affirmative or negative, and in that she could remember verbal promises. In considering the possibilities of her brain in this connection it must be remembered that no attempts were made to educate her. *Her hearing was acute,* and with the exception of the part played by the senses of smell and taste, her auditory sense seems to have been the link between herself and the outer world. It was by hearing and not touch that people were recognized; on hearing depended such powers of speech as she learned, and it was by hearing that she recognized what was going on around her, as shown, for instance, by her screams of anger when the furniture was moved preparatory to washing the floor, a proceeding associated in her mind with being disturbed from her usual seat.

(Vol. 27, 1904, No. 107, Autumn.)

1. On the Cells of the Spinal Ganglia and on the Relationship of Their Histological Structure to the Axonal Distribution. W. B. Warrington.
2. On Certain Tremors in Organic Cerebral Lesions. Gordon Holmes.
3. The Pathology of Infantile Paralysis (Acute Anterior Poliomyelitis. Frederick E. Batten.
4. So-Called Facial Hemihypertrophy. H. Mackay.

1. *The Cells of the Spinal Ganglia.*—The authors report the results and conclusions reached by a series of experimental lesions on cats. They review briefly the work of Doglei and others, who have showed that spinal ganglia contain not only the usual cell with the T-shaped axon of Ranvier, but others with differently distrubuted axons. The second cervical ganglion, which receives afferent fibers from the skin and small muscles, the eleventh dorsal, which also receives afferent fibers from the viscera; the seventh lumbar, which receives afferent fibers from the large hamstring muscles and has no visceral connections, were selected and 1,000 cells from each carefully measured, showing wide variations in size. Whereas in the dorsal and cervical region considerable similarity was noted in the proportion of cells of different size, in the lumbar ganglion appear a number of larger cells, and the authors conclude that these represent the cells in connection with the sensory organs, the muscle spindle of the large hamstring muscle. They further endeavor, following the work of Lugaro, Orr and Rows, to study by means of experimental section of nerve fibers of different distribution the axonal reaction occurring in the cells of corresponding ganglia, and to classify the types of cells thus identified according to the system of Lugaro. Chromatolytic changes were found occurring in the obscure cell and in the large clear cell, but the cell of the large granules, termed by the authors the coarsely granular cell, changed only when the section was made just beyond the ganglion, and the cells measuring less than 25 mmm. never showed any chromatolytic change. The paper is profusely illustrated, and the authors sum up as follows: (1) The size of the cells in the ganglia is a function of the diameter of the nerve fiber, and the difference of appearance in the chromophile elements has a dynamical significance. These elements represent material which is used up by the essential trophoplasm, which presides over the nutrition of the nerve fiber. (2) At the region of the limb plexuses a number of large cells appear in the ganglia, which are the cells of origin of the fibers to sensory muscle structures. (3) Lugaro's classification is confirmed in the main. The cells met with are best described as (*a*) clear cells, (*b*) obscure cells, (*c*) coarsely granular cells, (*d*) smallest clear cells. (4) The large clear cell and the obscure cell give origin to the fibers distributed to the skin, viscera and muscles. Large somatic and large splanchnic fibers have origin from cells of the same type, the large clear cell; small somatic and small splanchnic fibers also from cells of the same type, the obscure cell. (5) Afferent fibers to the thoracic viscera arise from the first five dorsal ganglia. The number of such fibers in one instance was found to be 136 in the first dorsal, 192 in the second white ramus. (6) The smallest cells in the ganglion, under 26 mikrons in diameter, whether belonging to the obscure or clear type, are immature cells, and have no fully developed axon. (7) The coarsely granular cell is highly resistant to the axonal reaction, and may be a "relay" cell, but sufficient evidence it not forthcoming as to its significance. (8) The cells in the early embryo have the appearance of the smallest type described in the text. In the newly-born human subject the types present in the adult can be recognized. The morphological characteristics of the cells of early embryonic life are also met with in the reactive phase.

2. *Certain Tremors in Organic Cerebral Lesions.*—The author first offers, as a substitute for the careless use of the word *tremor,* the following definition: "A clinical phenomenon consisting in the involuntary oscillation of any part of the body around any plane, such oscillations being either regular or irregular in rate and in amplitude, and due to the alternate action of groups of muscles and their antagonists." Reports are presented and discussed of nine cases in which tremor was a constant symptom, the rate being slow, varying from 3 to 5 oscillations a second, in all cases more or less regular in rate when limited to one group of muscles, in some cases absolutely so; and either regular or irregular in the range of amplitude. Some cases showed a periodical rhythmical increase and decrease of the range. In some instances the tremor showed a remarkable tendency to persist, apparently indefinitely, limited to the one movement if the position of the limb and other conditions remained unaltered, whereas in other cases it would involve various groups of muscles for short periods in succession, or even simultaneously, the resulting movement of the limbs being in the latter case compound and irregular, as the various oscillations were not synchronous in rate or equal in range. In every case the patient was unable to check the movement except for the shortest space of time, and it was often increased by inhibitory attempts. In addition to the tremor proper, volitional movement of the limb affected was in every case complicated by an irregularity of the intention-tremor type. The tremor never persisted during sleep, nor when the limb was at rest with every segment supported separately. An excited psychic state or forcible movements of the opposite limb had the effect of increasing the tremor. Other symptoms, not invariably present but associated with the symptom-complex, were: rigidity, paresis and uselessness of the limbs, affection of coördination and an emotional state. The rigidity was slight, rather a stiffness perhaps, never associated with organic contractures, constant throughout a movement, and associated with a marked slowness in performing and especially in initiating voluntary movement. The weakness seemed to be largely subjective, and best described by the term uselessness. The peculiar emotional states comprised a tendency to laugh or cry uncontrollably without adequate cause, dulness and apathy, sometimes accompanied by paresis of the expressional movements of the face. Other symptoms merely coincident with the symptom-complex are of interest as indicating the localization of the various lesions. In eight of the cases ocular palsies were a marked symptom: loss or defect of the vertical movements was frequently noted. Other symptoms to be grouped with these were a gait resembling that of paralysis agitans or cerebellar disease, slight hemianesthesia not of the cortical type, hemiplegic weakness with increased reflexes and extensor plantar response, and in two cases homonymous hemianopia on the side of the affected limbs. From a consideration of these symptoms the author reaches the conclusion that in each of the nine cases the focus of disease involved the dorsal portion of the mid-brain, and argues that involvement of the nucleus ruber, or at least of the cerebello-rubro-spinal system, is the essential factor in the lesion, and that destruction rather than irritation of these parts is responsible for the tremor. Consequently, an inhibitory influence on cortical action must be assumed for the cerebello-rubral system, whether normally exerted on the higher or lower motor centers, on the cortex or the spinal cord, the author does not find enough evidence to decide. Literature shows little attempt to classify the various disorders of movement, but the evident difference between the varieties would justify rigidly separating them in clinical study and assuming that they must be due to or constantly associated with lesions different in nature and site. From a study of sixty cases with autopsy collected from the literature the author concludes that athetosis almost always results from disease of the basal ganglia, but especially of the optic thalamus; that choreiform movements occur with about equal frequency in lesions of the

basal ganglia and of the mid-brain, whereas in the large majority of the cases of tremor the lesion involves the mid-brain.

3. *Pathology of Infantile Paralysis.*—The author notes the three views held as to the pathology of acute anterior poliomyelitis, namely: (1) That the condition is due to direct affection of the anterior horn cells; (2) that the condition is due to acute inflammation in the region of the anterior horns; (3) that the condition is due to a vascular lesion, which may be either thrombotic or embolic. Cases do occur belonging to the first group, but do not clinically conform to the usual type of the disease. The second view is the one most commonly held, but it is the third to which the author subscribes, and which he supports by the report and discussion of three cases. Head and Campbell have recently argued that the thrombosis is secondary to an acute infection, probably of bacterial origin, because experimental occlusion of spinal cord vessels is not followed by inflammatory change or hemorrhage. Batten answers with a reminder that obliteration of a large vessel is not necessarily comparable to the condition produced by thrombosis occurring in a smaller vessel, and recalls the experiments of Prevost and Cotard, who, by means of fine tobacco seeds, produced infarcts in various organs, and proved that congestion, hemorrhage and the exudation of cells were the early results of obliteration of small vessels. The fact that the area of softening corresponds so closely to the distribution of the branch of the anterior main artery makes the suggestion a reasonable one that such a condition is due to an occlusion of a vessel or vessels. Furthermore, we find the disease occurring most frequently in the lumbar region, a fact to be accounted for by remembering that the blood supply of the cord is at a point most distal from the heart, and that the reinforcing arteries have a very long course.

4. *So-called Facial Hemihypertrophy.*—The author reviews pretty thoroughly the literature on this rare condition, including reports of some thirty cases besides his own, that of a girl of ten suffering from acquired hypertrophy of the left side of face and skull. The child was normal at birth, and the deformity was first noticed at six years of age as a bony enlargement on the head near the vertex, followed by a similar one in the occipital region. Then came a fulness in the forehead, and lastly the cheek began to enlarge. The facial enlargement is limited strictly by the median line, and includes all the soft parts and the facial bones, together with most of the cranial bones. There is marked bossing of the frontal, parietal and occipital bones. The molars are longer and thicker on the left side, and the mastoid, malar and zygoma show enlargement. The posterior portion of the skull is also enlarged, and the hair covering it coarser than on the right. Tongue, palate and tonsils normal. The right eye has normal vision, but in the left the field is contracted to the nasal side. Hearing normal on right, markedly defective on left. Special senses otherwise normal. Sensibility to touch, pain and temperature unaffected. The author then gives a digest of the clinical features of the cases reported, followed by a discussion of the pathology and pathogeny. He thinks it not possible, either on clinical or biological grounds, to maintain the distinction indicated bv the words *congenital* and *acquired,* and would prefer to consider the morbid conditions in both classes to be the outcome of some disease process common to both, commencing prenatally in one class and postnatally in the other. Post-mortem observations of the condition are practically nonexistent. Portions of soft tissue removed during life show overgrowth of sebaceous and sudoriparous glands, and of adipose and connective tissues, with numerous degenerated epithelial cells. The views on the pathogeny of the condition are many and varied. Many writers take that of vasomotor paralysis, others that of circulatory stasis in the affected part due to vicious positions in the uterus, a theory which leaves the postnatal cases quite unaccounted for. Some attribute it to stimulation or irritation of epiphyseal cartilage and osteoplastic periosteum. A teratological theory has

been advanced, but this again fails to account for the postnatal cases. Finally, the author is inclined to favor the theory first advocated by Lewin, who gave a trophic rather than a vasomotor explanation of local atrophies and hypertrophies. The occurrence at times of hypertrophic dystrophies in syringomyelia and acromegaly add color to this theory. His own case is the only one in which occipital bossing has been noted, and this might be interpreted as pointing to a possible connection with acromegaly. The author thinks the following conclusions are warranted: (1) While nothing is known of the pathogeny of racial hemihypertrophy, the most reasonable explanation of the condition is that which associates it with disturbance of hypothetical nutritional or trophic centers in the perependymal gray matter of the encephalon. (2) In some of its features facial hemihypertrophy appears to have relationship with acromegaly and syringomyelia.

JELLIFFE.

PEDIATRICS.

(Vol. 16, 1904.)

1. Early Symptoms of Dementia Præcox. A. R. DIEFENDORF.
2. Cerebral Palsy of Childhood. M. L. GRAVES.
3. Natal Right Hemiplegia.
4. A Case of Hemiplegia Occurring During Whooping Cough and Diphtheria. HENRY FRASER.
5. Intraspinal Injections of Lysol in the Treatment of Cerebrospinal Meningitis. MORRIS MANGES.
6 Clinical Features of Cerebrospinal Meningitis of the Epidemic Type. HENRY KOPLIK.

1. *Early Symptoms of Dementia Præcox.*—Author speaks of the necessity of early recognition by the general practitioner. For convenience, groups the initial symptoms into four classes, namely, cases in which there is simple mental deterioration without hallucination or delusions; cases in which a few indefinite delusions appear; cases of a neurasthenic character; cases of hysteroid type.

2. *Cerebral Palsy of Childhood.*—Postnatal left hemiplegia. Occurred at eight years of age, during an attack of typhoid. At twenty years admitted to hospital because of mental impairment. No history of convulsive seizures.

3. *Natal Right Hemiplegia.*—Tedious birth, no instruments used. Fretful immediately following birth. Paralysis of right side noticed at three months of age. Epileptic convulsions at twenty months. At eight years mind normal; fair scholar. Abandoned school attendance because of frequency of convulsions. At sixteen committed to hospital because of impaired mental state. At nineteen general condition grave, mind nearly extinct.

4. *A Case of Hemiplegia Occurring During Whooping Cough and Diphtheria.*—Male child of eighteen months, ill with diphtheria and whooping cough. Antitoxin used. On sixth day of diphtheria had convulsion, followed by paralysis of right side. Right hemianesthesia. Later, convulsions of Jacksonian type, right lateral hemianopsia. Hemiplegia thought to be due to intracranial hemorrhage.

5. *Intraspinal Infections of Lysol in the Treatment of Cerebrospinal Meningitis.*—Three cases reported successfully treated by lumbar puncture and lysol injections. after a modification of the method of Seager, of Lisbon.

6. *Clinical Features of Cerebrospinal Meningitis, or Cerebrospinal Fever of the Epidemic Type.*—Paper very interesting, complete as to detail, etc. The disease, like pneumonia. considered to be self-limited. At present the most to be expected of treatment is to relieve suffering and prevent complications. Lumbar puncture cannot be considered as curative, although many consider it a procedure attended with decided benefit. Thirty-one cases treated by Seager (lumbar puncture and lysol injection) show a

mortality of 42 per cent. Twenty cases treated by same person by means of lumbar puncture show a mortality of 43 per cent.

J. E. Clarke (New York).

MONATSSCHRIFT FUR PSYCHIATRIE UND NEUROLOGIE

(Vol. 16, 1904, No. 5, November.)

1. The Application of the Ergograph in the Investigation of Ataxia, with Other Ergographic Results in Diseases of the Nerves. Aub.
2. The Population of the Netherlandish Insane Asylums in the Second Half of the Nineteenth Century. W. Schermers.
3. Delirious Asymboly and Epileptic Oligophasia. A. Bernstein.
4. Therapeutic Results in Acute Psychoses. F. Klein.
5. The Psychoses of Prisons. N. Skliar.
6. Compensatory Movements in Affections of the Brain. W. v. Bechterew.
7. The Seventy-sixth Assembly of German Naturalists and Physicians in Breslau, Sept. 18 to 24, 1904.

1. *Ergograph in Ataxia.*—Aub has employed the ergograph of Kræpelin, which is so arranged as to show the smallest possible error. Nevertheless, the instrument still records complicated muscular contractions, and not the contraction of a single muscle. Aub describes the application of the instrument, and certain precautions that must be taken in its use.. His results were as follows: In tabes there is a slight diminution in the fatigue.. Even the fourth or fifth successive curve shows little alteration, although normally there is a distinct change in the second curve. There is more variability in the curve than in the normal cases, and this is increased by closing the eyes. In Friedreich's disease the curve is irregular, and this irregularity is not increased by closing the eyes. In multiple sclerosis the patient rapidly becomes fatigued; the curve is even more irregular than in tabes, and this irregularity is not increased by closing the eyes. In cerebellar disease the irregularity of the curve is not particularly marked. In cortical ataxia the disturbance or unilateral disturbance is very clearly shown. In peripheral paralysis the elevation is low and frequent, corresponding to the degree of paralysis. In paralysis or paresis due to lesion of the pyramidal tracts the elevation of the curve is lower than normal and the frequency of the elevation is less. Certain variations in the character of the curve could also be made out. Some excellent illustrations accompany this article.

2. *Insanity in the Netherlands.*—Schermers shows that the number of inmates of the insane asylums in the Netherlands has increased not only absolutely but relatively to the increase in the population in the last fifty years. The more chronic forms of insanity seem to be common, because the percentages of recoveries and of deaths have both slightly diminished. He believes that some method of getting rid of the chronic cases should be adopted, either colonization or distribution among families. Otherwise insane asylums are more likely to become homes than hospitals for the cure of disease. Moreover, the asylums are so crowded that it is difficult to find places for recent cases, and they are not admitted until the disease has reached an incurable stage.

3. *Asymboly.*—A woman of 25 with acute pneumonia developed acute dementia. As a characteristic symptom there was disturbance of the recognition of objects. She was unable to name accurately various pictures of familiar things. This Bernstein calls asymboly. It occurs particularly in delirium tremens. The second case, a woman of 52, suffering from a form of hysteria common in Russia and known under the name of "klikuschestwo," when shown a picture was only able to recognize it after she had traced its outlines with her finger. Very small pictures, however, she could recognize at once. Bernstein believes that this symptom is due

to extreme contraction of the field of vision. The existence of a contraction could also be determined. The third case, a boy of 19, had epilepsy and was arrested and found in a state of confusion. He appeared incapable of recognizing any object. Later he recovered. There appeared to be a double symptom here: inability to recognize, and later, inability to name correctly the objects. The latter symptom may be considered a functional aphasia or oligophasia. Bernstein discusses the literature of the transient disturbances of speech in epilepsy.

4. *Treatment of Acute Psychoses.*—Chloral-hydrate is a valuable hypnotic, acting particularly well in maniacal conditions, in doses of from 20 to 30 grains. It is contraindicated in all forms of organic heart disease, atheroma, chronic nephritis and conditions that interfere with respiration. It should not be employed for long periods, because it gives rise to gastritis, ulcerative enteritis and scorbutic diseases. The combination of morphium with chloral-hydrate should not be employed. Paraldehyde is practically without danger. Occasionally, if the patient does not sleep there is a light delirium with slight reddening of the face. Even large doses are not dangerous, and one dram may be given two or three times in the course of an evening. It has a disagreeable odor and taste, produces a disagreeable odor on the breath, and if its use is continued it causes loss of appetite and its action is not sedative. Dormiol, a mixture of chloral-hydrate and amylhydrate, is less valuable than either chloral or paraldehyde. It may produce gastrointestinal catarrh, congestion of the head, and occasionally exanthemata. Amylanhydrate is chiefly used in status epilepticus or states of hysterical confusion. It should be given into the muscles. In the general treatment of acute psychoses Klein believes that there are four important factors: First, rapid quieting; second, conservation of the vital force; third, improved supervision; and fourth, a more exact observation of the vegetative functions. Hydrotherapy is a most valuable measure. Prolonged full baths are of great value in maniacal conditions, or in states of hallucination with ideas of grandeur and motor excitement. A more intermittent form is useful in cases of precordial distress, in climacteric psychoses and in hystero-neurasthenic conditions. In states of meloncholia prolonged full baths are of disadvantage. Sometimes prolonged bathing produces a diminution in the weight of the body. Fresh air is also a calming force in states of excitement. A rich, stimulating diet is also of importance. When patients are first admitted it is Klein's custom to administer an adequate dose of castor oil emulsified in hot milk. Alcohol can be used with advantage, particularly in the form of red wine. It stimulates the appetite, prevents collapse and is particularly valuable in old age. Occasionally it is necessary to use the stomach tube or to nourish the patient by nutrient clysmata. It is important that the patient should receive an adequate supply of fresh air. Finally, it is important to keep the patients occupied, even in states of acute psychosis.

5. *Prison Psychoses.*—Skliar continues his description of cases of insanity occurring among the prisoners in Switzerland. Among the symptoms hallucinations were common. The disease develops rapidly and recovery is usually prompt. If, however, the patients are maintained as prisoners after the occurrence of the symptoms it is not uncommon for dementia to follow the acute attack. The imprisonment itself must be regarded as the most important determining cause.

6. *Compensatory Movements in Hemiplegia.*—Von Bechterew describes two cases in which compensatory movements occurred in the opposite side in the course of hemiplegia. He believes that the only explanation is the transference of the irritation to the other sphere.

7. *Breslau Meeting.*—The papers read at the meeting of the German naturalists and physicians in Breslau, Sept. 18 to 25, 1904, were as follows: Amentia, by E. Stransky; Clinical and Anatomical Contribution to the Pathology of the Left Temporal Lobe. By C. Freund; A Clinical Contribu-

tion to the Pathology of the Left Temporal Lobe, by Freund; A Case of Choreatic Disturbance of Motion in an Adult, by Köbisch; Investigations upon the Cerebrospinal Fluid, by A. Fuchs; The Method of Cytologic Investigations, by R. Rosenthal; Hypnosis Hysteria; their Common and Opposed Relations in the Light of Mechanistic Universal Consideration, By Grossmann; The Metabolism in Obstinate Cases of Mental Disease, by Rosenfeld; The Influence of Cerebral Moments upon Menstruation, and the Treatment of Disturbance of Menstruation by Hypnotic Suggestion, by Delius; Röntgen Pictures of the Head in Disease of the Base of the Skull, by Schüller; Report upon the Pathogenesis of Choked Disc; by Saenger.

(Vol. 16, 1904, No. 6, December.)

1. The Precocious Form of Dementia Paralytica. G. Eisath.
2. The Significance of the Babinski Plantar Reflex, and the Oppenheim Leg Reflex as a Criterion of Disease of the Pyramidal Tracts. B. Pfeifer.
3. A Case of Dementia Paralytica Following Abdominal Typhoid, with Termination in Complete Recovery. O. Foerster.
4. A New Theory of Hemiplegic Disturbance of Emotion. M. Rothmann.

1. *Precocious Dementia Paralytica.*—A girl whose father had been alcoholic, but apparently never syphilitic, and who had not been exposed to infection, at the age of 23 had an attack during which she slept for fourteen days. Immediately afterward she became gay and had delusions of grandeur, with, from time to time, periods of excitement. The pupils reacted well; there were no disturbances in any of the cranial nerves and no disease of any of the viscera. Her condition varied from delusions of grandeur, excitement almost maniacal in character, stuporous stare and an almost normal condition, in which she realized that her mind was affected. Later she improved, returned home, developed an ulcer of the stomach, grew worse and was returned to the asylum, where it was found that the pupillary reactions were no longer normal. She had an attack resembling uremia (though there was no albumin in the urine), during which she died. The microscopical examination showed changes resembling those of dementia paralytica. Discussing this case, Eisath calls attention to the frequency with which hereditary syphilis, or syphilis acquired in youth, is the cause of an early form of paralytic dementia. He believes the severity of this case was due partly to the fact that there is no reason to suspect previous syphilitic infection, partly to the fact that the paralytic symptoms developed so late.

2. *Babinski and Oppenheim Reflexes.*—Oppenheim's reflex consists of a dorsal flexion of the toes, if the inner side of the leg just back of the edge of the tibia is stroked with a blunt object. It occurs only in patients with the spastic symptom-complex. In normal individuals this stroking usually produces a plantar flexion of the toes. In patients with a hypotonic symptom-complex it is more frequently absent than in normal individuals. It is less constant than the Babinski reflex, which in other respects it resembles. Pfeifer has examined this reflex in a number of cases, comparing it with the plantar reflex. In a case of infantile convulsions it was present, although the Babinski reflex was absent. In fourteen cases of multiple sclerosis the results differed in nine. He also discusses a number of other conditions in which there was difference in the reflexes, and is unable to find any satisfactory explanation of this. In regard to the occurrence of both reflexes, he supposes that there are bulbar or cerebello-spinal tracts which are only active in the earliest periods of the development of the spinal cord. These produce dorsal flexion upon the sensory stimulation of

the skin of the foot, the arc being completed in the cortex. Later the centrifugal fibers passing through the pyramidal tracts produce plantar flexion upon the stimulation of the skin. If the conductivity of the pyramidal tract is impaired or destroyed the tracts of earlier development resume their function and produce the dorsal flexion.

3. *Dementia Paralytica and Typhoid.*—A man of 42, without syphilitic history, had a severe attack of typhoid fever. When he was able to leave his bed it was noted that his character had changed. He took interest in nothing; was careless about himself; complained of headache; slept little, and was sensitive to sounds. Later he complained of vertigo and ceaseless tinnitus. His speech was drawling; there was tremor of the muscles of the face and hands, but the pupils were normal. There was hypotonia of the legs, patellar clonus and increased Achilles tendon reflexes. His intelligence was greatly diminished. His general condition was that of apathy. He gradually recovered and in the course of nine months was apparently perfectly normal and capable of returning to his work, which he performed satisfactorily. Foerster regards it as a case of post-typhoid disease of the central nervous system.

4. *Emotion and Hemiplegia.*—Rothmann believes that, in addition to the pyramidal tracts, there are other tracts in the spinal cord conveying impulses centrifugally. Whether these tracts are in a state of functional activity when the pyramidal tract is normal is a question that cannot be determined, but there is some evidence which shows that they are. When the pyramidal tract is destroyed it is possible that they become active. Complete relaxed paralysis occurs when not only the cortical but the subcortical centers are destroyed. If only the cortical centers are destroyed then the subcortical centers may become active and account for the hemiplegic disturbances of movement. J. Sailer (Philadelphia).

REVUE DE PSYCHIATRIE ET DE PSYCHOLOGIE EXPERIMENTALE

(January, 1905.)

1. Latent Uterine Infections in the Newly Delivered and Their Importance in Mental Medicine.—Visceral Insanity. Picqué.
2. Vesanic Dementia. Is it a Dementia? Toulouse and Damaye.
3. Some Facts on the Mental Improvement Following Fever. Vaschide.
4. A Case of Abstraction in a Dog. Piéron.
5. The Metatrophic Treatment of Epilepsy. Kinberg.

1. *Uterine Infections.* In this review of the phychoses of the postpartum period the acute septic infections are not considered. Fourteen cases are cited and discussed. All of these cases were surgically treated, with the result that six recovered, three improved and five were unimproved. The article is a plea for systematic gynecological examination of insane women and surgical intervention when indicated.

2. *Vesanic Dementia.*—Continued article. Will be abstracted when concluded.

3. *Mental Improvement following Fever.*—Three cases are cited, one of maniacal excitement, one of depression and one of mystical delirium. The first two had attacks of la grippe, the third of pneumonia. In each instance the mental condition cleared up during the fever, the symptoms returning when it subsided.

4. *Abstraction in a Dog.*—Of no interest to the alienist.

5. *Treatment of Epilepsy.*—The method of treatment employed is the method of hypochlorization with the administration of sodium bromide. The conclusions reached are as follows:

1. The treatment diminishes to a considerable degree the frequency and the intensity of the attacks, and appears to prevent completely status epilepticus.

2. It often exercises a happy influence on the habitual psychic state and prevents the clouded and delirious states.

3. It influences oftentimes the symptoms of intoxication, caused probably in part by the lack of sodium chloride. That fact indicates some cases when the method ought not to be applied:

- *a.* Affections of the heart.
- *b.* All patients with cardiac fatigue from special causes (nephritis. emphysema, etc.).
- *c.* Obesity (even acquired during treatment), cases when affections of the heart are imminent.

(February, 1905.)

1. Researches on the "Yellow Pigment" of the Nerve Cells. MARINESCO.
2. Vesanic Dementia. Is it a Dementia? TOULOUSE and DAMAYE.
3. Animal Psychology. HENRY PIÉRON.
4. The Prophylaxis of Relapses in Mental Medicine.

1. *The Yellow Pigment of Nerve Cells.*—The article begins with a historical review of the researches on this yellow pigment and the opinions founded thereon. The author thinks the time opportune for a critical study with the aid of the new methods of histology. Among these new methods three appeal to him as especially valuable: The Sudan stain, the method of Marchi, or rather of Buxh, and the new nitrate of silver method of Cajal.

The factors that determine the appearance of pigment, and above all of yellow pigment, are numerous. Here are the principal:

1. Age. The premature appearance of yellow pigment is a sign of precocious senility.

2. Nutritive disorders of the cell, such as progressive anemia of the nerve centers, the slow intoxications, nerve sections not followed by repair, and various degenerations which hinder the repair of the chromatophilic elements.

3. The formation of pigment accompanies all states of pathological degeneration.

The author does not think the yellow pigment is fat or a body allied to it, as supposed by Obersteiner. The yellow transformation is slow, while fatty degeneration is an acute process, such as observed in phosphorous poisoning. Mülhmann believes also that it is of a fatty nature, and Rothmann and Ohner classify it as lipchrome. This is based largely on the reactions it has in common with myélin. The author admits that the pigment contains lecithin, a body always accompanying fat, but does not think it a lipochrome; it does not show the chemical reaction of lecithin.

Practically nothing is known of the function of the yellow pigment, while two theories have been advanced as to its origin: 1. It appears in the fundamental amorphous substance of the cell as the product of cellular activity, either of a regressive nature, that is to say, resulting from decomposition, or from the increase in the albuminoids found there. 2. It results from the disintegration of the chromotophilic substance or the destruction of the neuro-fibrils.

Yellow pigment thus constitutes a normal product in the lives of certain cells, while in certain pathological conditions the quantity of pigment assumes considerable proportions, the limits between normal pigmentation and pigmentary degeneration are not easy to trace.

2. *Vesanic Dementia.*—It has for a long time been usual to speak of the terminal conditions of the vesanias, such as the manias, melancholias, persecuted, hypochondriacs, in which after a long course incoherence supervenes, with perhaps automatic gestures and untidiness as dementia. An examination of some of these cases has shown, however, that the memory is more profoundly disordered than diminished. Early writers vaguely saw a difference between the dementia terminating the organic affections of

the brain and that terminating the vesanias, particularly Baillarger, who described these two categories, and Griesinger, who writes of the latter, "The ideas are dissociated and the signs in disaccord with the ideas; but the intelligence is not abolished, and the exercise of the intellect is quite active."

Many vesanic dements in the midst of their incoherence give correct answers to questions, while at other times the answers are erroneous. This proves that the knowledge is present all the time, and it seems more logical to avoid the name of the dementia, which indicates a permanent, destructive, organic change in the brain, and call these cases of confusion.

Confusion often complicates the picture in paresis and makes the dementia appear much more profound, and Régis believes that many of the cases of dementia præcox, particularly those that recover, are cases of confusion—pseudo-dementia præcox.

For the purpose of demonstrating whether there really is a distinction between organic dementia and confusion the authors have devised some clinical tests. These tests comprise five series of simple questions in geography, history, simple calculations, and terminating with an easy syllogism to test the judgment. The ordinary school knowledge is avoided. The examination is conducted three times in each case and the results studied.

The classes of cases were studied—paretics, vesanics and aged vesanics constituting an intermediate grade. As a result of these studies the authors do not attempt to erect a law, but simply to emphasize the distinction of the two classes by showing the minimal intellectual enfeeblement of the vesanics and the preponderance among them of a state of confusion.

3. *Animal Psychology.*—Of no interest to psychiatrists.

4. *Prophylaxis of Relapses.*—This is a page quoted from Erquirol's "Des Maladies Mentales." In it this noted alienist lays great stress that relapses are often preceded by moral and physical disorders, and he calls attention to the great necessity that confronts the person who has suffered from an attack of insanity of avoiding all such exciting causes.

(March, 1905.)

1. Researches in Pedagogic Psychology. Henri Piéron.
2. Predisposition and Direct Causes in Mental Etiology. E. Maraudon de Montyel.
3. New Method of Ramón y Cajal for Staining Myelin Fibers. Marchaud.

1. *Pedagogic Psychology.*—A review largely of exerimental studies on school children.

2. *Predisposing and Exciting Causes.*—The author does not believe that an exciting cause alone is enough to produce a psychosis—the soil must have been prepared. In closing, the author speaks of certain exciting causes of mental disease and the fact that they imprint their peculiar characteristics upon the attack. Alcohol and epilepsy are examples of such, and he thinks typhoid and Parkinson's disease also produce a distinctive mental symptom-complex when insanity results.

3. *New Method of Staining Myelin Fibers.*—This article is purely technical—describes a detailed process in its different stages.

W. A. White (Washington, D. C.).

MISCELLANY

Extirpation of the Gasserian Ganglia. Harvey Cushing (Journal A. M. A., March 11, 18, 25 and April 1 and 8).

The author discusses various operative methods for dealing with the ganglion in cases of major trigeminal neuralgia. The importance of total removal of the ganglion he considers is evident, and were the anatomic difficulties less it would be the only method free from criticism. This method alone, according to our present knowledge, insures total and permanent freedom from pain. He gives details in full of his method, which is different from that of Lexer, subsequently published and similarly successful, in the management of the meningeal artery, in that Cushing, so far as possible, carefully avoids injury to this artery, and generally with success. He also favors removal of the zygoma for cosmetic purposes, the slight flattening of the face thus produced being less disfiguring than the prominence of the bone with the muscular atrophy that follows the operation. He has also been able in many of his cases to reach the ganglion without much loss of blood, and claims that the small opening made by his method affords advantages in this regard during the removal of the ganglion itself and lessens the amount of packing required. He has had little success with the use of adrenalin or other methods than packing in checking the hemorrhage. Blood-pressure observations taken before and during the use of anesthetics are valuable in this connection as showing the patient's condition, and Cushing thinks that possibly his one fatal case might have been saved had more attention been given to the warnings furnished by this procedure. The clinical histories of his twenty cases thus operated on are given in detail and the results summarized. He remarks that he has not in all cases followed Krause's dictum that the ganglion operation should be performed only in those cases where previous neurectomies have been given a trial. In well-developed major neuralgia he thinks temporizing inadvisable, even for the temporary relief it may afford. The most important of the post-operative complications are those of the eye, and it is well to have an understanding with regard to this before operation. The commonest sequel is injury to the abducens. For safeguarding the eye Cushing uses rubber protective, and after the operation he advises the use of a large watch glass. with the edges covered with adhesive plaster so as to exclude the air and continued as long as conjunctival irritation threatens. His paper concludes with a number of physiologic considerations on the functions of the nerves and some remarks as regards the pathology. The morbid lesions underlying true trigeminal neuralgia are not yet revealed. The paper is fully illustrated.

IS DELUSIONAL INSANITY DUE TO DISEASE OF THE BRAIN? J. W. Wherry (The Alienist and Neurologist, February, 1904).

In the classification of insanity the author, according to origin being due to disease of the brain or otherwise, divides mental disorders into two classes: (1) Imbecility, general paresis, dementias. (2) All other forms of insanity grouped under general term of delusional insanity. With this distinction, prognosis is also considered. Those of the first group, having origin in disease of the brain, are incurable. Those of the second group, having origin in disturbed visceral function, are mostly curable. From pathological, physiological and psychological data, the writer discusses correctness of his opinions that "delusional insanity" is not of cerebral origin. J. E. CLARK (New York).

THE GLOBUS HYSTERICUS AND ESOPHAGEAL SPASM. Buch (Gazetta d. Osp. e d. Clin., March 16, 1905).

It is generally accepted that globus hystericus is a spasm of the esophagus, but Buch calls attention to the fact that in spasm of the esophagus there always exists difficulty of deglutition, which is never the case in hysteria. The esophageal spasm is accompanied by a painful sensation of tightness, by a real pain, but the patient does not experience the sensation conveyed by the pressure of a foreign body in the esophagus; it is otherwise with the hysterical subject; while the sensation of pain is absent, the patient complains of a foreign body in the throat. The esophageal spasm attacks one definite spot, which may vary on different occasions, but never during the attack, the point most frequently involved being directly behind the sternum, whereas the globus hystericus is rather movable, often changing its place several times during one attack. Once the globus reaches the throat there is at once a sense of suffocation, which is never present in esophageal spasm, nor is there any vomiting in hysteria. Physiological contractions of the organs in the throat cause usually no sensation, whereas spastic contraction may sometimes be accompanied with pain; but never calls out the sensation of a foreign body. It should also be remembered that globus hystericus may first originate in the lower region of the abdomen, and thence travel upward. It thus becomes clear that we really have to deal, not with a cramp, but with the sensation simulating one. It is evidently the sympathetic nerve which may serve as the organ of irradiation, thus conveying to the patient the sense of pain in a locality different from the one where it really originates. On examining twenty hysterical patients with reference to the point the author obtained the following results: There was in all the cases a marked hyperalgesia of both the lumbar and the cervical sympathetic; this hyperalgesia was associated with real pain in the nerves that radiate from these regions; in almost half of the cases the globus hystericus could be called out by pressure on the lumbar part of the sympathetic. The globus hystericus consists of a sensation that emanates from the terminal filaments of the sympathetic with the included prevertebral branches. It thus happens that the sensation experienced by the hyperalgesic lumbar portion of the sympathetic tends to affect the hyperalgesic ganglia of the terminal nerves in such a way that the successive repetition of the stimuli determines in the ganglia themselves an activity which would otherwise remain latent. ROVINSKY.

EXHAUSTION DISEASES OF THE NERVOUS SYSTEM. L. Edinger (Deutsche med. Wochenschrift, 1904-1905).

Edinger, in an interesting series of articles, discusses once again the theory which he advanced some years ago that there are nervous diseases which develop because, under certain circumstances, the normal demands imposed by the performance of function are not met by a corresponding restoration within the tissues. The characteristic of this condition is a simple atrophy of nerve fiber. All diseases of the nervous system can be divided into focal diseases, toxic affections and exhaustion diseases. In healthy persons exhaustion is characterized anatomically within the cell by a disappearance of the tigroid bodies and perceptible changes in the medullated fibers. If the using up of tissue is too great, or the replacement is insufficient, there is more permanent and complete destruction of the cell and fiber. The glia proliferates to take the place of the degenerated nerve tissue. The process in all these diseases is identical, but the location differs. They are all progressive. These exhaustion diseases may arise on account of abnormal demands on the normal tracts, although there is normal restoration of the tissue. In this way may be explained the various atrophies from disuse and the professional neuritides. In other cases there is not a sufficient restoration to meet the demands of normal functions. These cases are usually due to some poison, such as syphilis or lead. He gives a number of interesting observations of lead paralysis to prove that the paralysis first affects the muscles upon which the greatest demands are made, and shows the excessive use of the extensors of the wrist in the use of the paint brush. He gives many instances also of the effect of over-exertion in one part or another in exciting or aggravating certain special symptoms of tabes. The cause of the exhaustion may differ according to the nature of the poison. Examples of this second class of exhaustion diseases are polyneuritis, tabes, combined systemic diseases and general paralysis. Exhaustion diseases may also develop when different nerve tracts are from the outset not sufficiently developed to be able permanently to perform their functions, but atrophy prematurely—the abiotrophy of Gowers. The hereditary nervous diseases, many of the combined scleroses, amyotrophic diseases of the cord and medulla, primary nontabetic optic atrophy, and probably progressive nervous deafness, belong to this class. Several nerve tracts may be affected in these conditions, either simultaneously or successively, and various combinations may occur. The types are not always constant; thus, tabes and general paralysis may often co-exist, and tabes and spinal amyotrophy sometimes occur together. Combinations in the third group, however, are not observed, because here some definite tract is congenitally defective while other regions are normal, and thus this tract becomes more easily exhausted; whereas poisons exert an influence upon the entire nervous system, and that part is affected which is most used. The old theory of a selective action of poisons, such as the selective action of lead upon the peripheral motor neurones to the extensors of the wrist, is regarded as no longer tenable; those neurones are affected because they are most exhausted by the demands put upon them. The therapeutic applications of this theory are obvious. With a given predisposition, toxic or hereditary, the possibility of exhaustion must be borne in mind. Thus tabetic patients should walk little, take only such exercise as does not fatigue, urinate every hour, wear dark glasses in bright sunlight, go to bed for a few days at any exacerbation of the disease, and fear any strain. By this means of treatment Edinger states that of late, for example, he has observed no vesical paralysis in tabes. In multiple neuritis absolute rest in bed is requisite; in mononeuritis fixation of the part by splints. By careful application of this theory Edinger believes that we shall obtain greater results both in treatment and prophylaxis. (The theory thus advanced with a wealth of illustration and argument, for which space is lacking here, is interesting and suggestive, but it hardly seems absolute. In point of fact, in tabes, for example, we still know very little as to the determining cause. Syphilis is most probably the chief, perhaps the sole, predisposing cause, but we know nothing as to why one syphilitic becomes tabetic and another remains well nor, even though Edinger finds a history of exhaustion as the exciting cause in some cases of tabes, was there a greater amount of exhaustion than with other syphilitics who did not become tabetic. Or, to take one individual symptom of tabes, optic atrophy, the optic nerve is constantly stimulated during our waking hours more than any other sensory nerve of the body. Why should not optic atrophy be the first and most constant symptom in tabes, instead of being observed in only a small percentage of the cases? Why, furthermore, should exhaustion affect the sensory neurones under the influence of syphilitic toxin, and the motor neurones under the influence of lead? Even on the hypothesis that to the exhaustion is added a congenital defect of certain nerve tracts, sensory or motor, lead tabes and syphilitic amyatrophy ought to be commoner affections. Nevertheless the theory, although not fully adequate, is suggestive, and the therapeutic indications are of much promise. Certain of them, indeed, as rest in bed for tabetics and immobilization by a splint in mononeuritis, have long been recognized as beneficial.) Philip Coombs Knapp, in *Boston Medical and Surgical Journal.*

Book Reviews

ANNUAL REPORT OF THE GOVERNMENT HOSPITAL FOR THE INSANE, Sept. 15, 1904.

This is the forty-ninth annual report of the hospital, and the second communication of the present Superintendent, Dr. Wm. A. White. The first section, relating to the material side of the institution, is of more than usual interest, as it records the completion and partial occupancy of fifteen new buildings, which cost about $1,500,000. It also shows evidence of a general renovation and improvement of the physical basis of almost the entire institution. Among many other new departures we note the boring of additional artesian wells, the installation of an apparatus for pasteurizing milk, the remodelling of the laundry so that infected clothing can be sterilized, the increased fire protection by fireproof stairways, fire escapes and an automatic fire alarm system, the enlargement of the laboratory, and the introduction of a complete system of card indexes for cases and filing cabinets for case records.

The section on medical work shows that this widespread activity has already produced good results. From the records of the pathological department it appears that while typhoid fever during the past year has been unusually prevalent in the District of Columbia, not a single case has occurred at the hospital in a general population of nearly 4,000 persons, whose water supply has come exclusively from artesian wells. In the previous year, when a certain amount of hydrant water was used, 29 cases occurred, with three deaths.

In regard to malaria, a report of 1896 is quoted to the effect that 47 per cent. of the population were treated for intermittent fever. Last year only two cases are recorded. and this reduction is attributed to the drainage of surface water, the abolition of cesspools and the more liberal use of window screens

Tuberculosis has been cut down from an average of 22.9 per cent. for the last 20 years to 13.6 per cent. by the segregation of over half of the tubercular patients by the building of a solarium, and probably in part by condemning the cattle of the hospital herd which react to tuberculin.

The pathological department also reports 103 autopsies from the 245 deaths, and the addition of 25 specimens and numerous photographs to the museum. It is noteworthy that 23 per cent. of the cases of pulmonary tuberculosis among the insane showed secondary intestinal involvement. Among the cases specially mentioned were 33 cases of softening or atrophy of the brain secondary to arteriosclerosis, two cases of dural hematoma, one case of gummata of the brain, one of rupture of the coronary artery with hemopericardium, one case of abscess of the heart wall, four cases of aneurysm, including one of the heart, and five cases of cancer of the stomach, four of which showed metastases.

In the way of recommendation Dr. White protests against the present method of committing civil patients from an open court like a criminal, and emphasizes the need of recognizing insanity as a form of illness. Along the same line he suggests that the objectionable term insane be omitted from the official title of the institution. Successful staff meetings have been held weekly for the discussion of journal articles and case histories, but the Superintendent believes that a more thorough organization of clinical work is indicated.

Then follow elaborate statistical tables in regard to admissions and discharges. nativity, causes of death, form of mental disease, etc. Several tables include data from the opening of the hospital in 1855, when the total admissions were 63 patients.

"During the year ended June 30, 1904, there were admitted to the hospital 766 patients. Of this number 270 were from the Army, Navy and Marine Hospital Service, while the remaining 496 were from civil life.

"The total number of discharges for the same period was 643, classified as follows: Recovered, 235; improved, 130; unimproved, 34; not insane, 1; died, 243. The total number of patients under treatment during the year was 3,135, there remaining under treatment on June 30, 1904, 2,492. These figures show an increase of 123 patients over the number for the past fiscal year."

In the body of the report are found six handsome cuts of new buildings, and the volume is closed with a detailed statement to Congress of the year's expenditures. JELLIFFE.

THE DOCTOR'S RECREATION SERIES. VOLUME IV. A BOOK ABOUT DOCTORS By JOHN CORDY JEAFFRESON. The Saalfield Publishing Company, New York, Chicago, and Akron, Ohio.

A fourth volume of "The Doctor's Recreation Series" brings us further diversion in the shape of researches into the curious annals of the past by an author whose talent for such a task has already been demonstrated in his previous work, "A Book About Lawyers." Old days and old ways never fail of interest for the modern mind, and the chronicles of the medical profession are peculiarly rich in curious and instructive, as well as amusing matter, owing to its invasion by all sorts of superstitions and crudities, and to the eccentric character of many of its votaries in the earlier days. Mr. Jeaffreson has skillfully selected and aptly arranged from this mass of material that which is best calculated to entertain, and doctors of the old school from the time of wig and stick appear most vividly in his pages. Yet in spite of the large rôle played by credulity, ignorance and eccentricity in the varied tales of bygone days, the true, unselfish enthusiasm and devotion of some of these men who must meet obstacles undreamed of by the modern physician cannot but increase the reader's respect for this much-enduring profession. GOODALE.

A HISTORY OF SCIENCE. By HENRY SMITH WILLIAMS, M.D., LL.D., assisted by EDWARD H. WILLIAMS, M.D. In five volumes, illustrated. Harper and Brothers, New York.

To write the history of science, to tell the storv of "organized knowledge" from the cave dweller to Marconi and Curie, from scraped bone to radium, is doubtless a stupendous task, but a fascinating one as well, both to author and to reader, and it would be hard to tell which part of the story, which one of the five substantial volumes, deserves that epithet most markedly.

The beginnings of things always have a charm of their own, and Dr. Williams has devoted a volume to the beginnings of science, from the prehistoric period up to the time of Galen. Until that time the mass of human knowledge was subject to little classification, and the story can proceed in regular chronological order, embracing in the progress of science philosophy, medicine, mathematics, astronomy and whatever else the race had as yet acquired as a basis for later developments. If the author's treatment of this period did no more than impress upon the reader the essential unity of science, the continuity which includes the first crude gropings and the latest complex achievements it would still be amply justified. But it does more. The graceful, vivid handling of material which is characteristic of the whole work is already evident in this volume. To be sure, the material itself is an inspiration. As the author himself says: "Nothing but dulness in the telling could mar the story," and he has been peculiarly successful in keeping the "human interest" strong in its appeal. One factor which has contributed to this success is his handling of the personality of the men who made the history which he records. Names to conjure with he has in abundance, and from Thales to Haeckel he quite legitimately makes the most of their glamour. But when his reader, lured by the "Homeric catalogue" of names that echo down the ages, takes up these volumes he finds by the time he puts them down that his vague impressions have changed to definite knowledge of what these men stood for and how each one set his stone in the mighty structure of world wisdom. The general public will welcome the nice balance of the book between technical incomprehensibility and the inaccuracy which is the bane of so-called "popular" works on science.

The second volume takes up the work of the middle ages with no "dulness in the telling" of that gallant struggle with reactionary forces, with the pseudo-sciences of astrology and alchemy, and all the bitter opposition that gives such men as Roger Bacon and Galileo something of the martyr's halo. Paracelsus, Harvey, Leibnitz, Newton, Franklin and Linnæus bring the tale to the close of the second volume and down to the dawn of modern science.

But now the stream has become too broad for simple chronological sequence, and the author follows separately each branch of science to some convenient halting place from which he turns back to bring up in turn the other branches.

Volume III is devoted to the modern development of the chemical and biological sciences, and Volume IV to that of the physical sciences. Space forbids even the baldest enumeration of the men and the achievements treated of, and we can only say that the passing of the charm of remote ages in no way hampers the author's hold upon the reader. The comparatively swift development and differentiation of modern science with all its magic and its precision is told with no lack of grace, and even a touch of fancy now and then, as when in the chapter on geology he says of the Rocky Mountains: "High and erect these young mountains stand to this day, their sharp angles and rocky contours vouching for their youth in striking contrast with the shrunken forms of the old Adirondacks, whose lowered heads and rounded shoulders attest the weight of ages."

The final volume sums up the aspects of recent science, presents some of the unsolved problems, and closes with an optimistic outlook.: "Our leaders of thought have struggled free from the bogs of superstition, and are pressing forward calmly, yet with exultation, toward the heights."

JELLIFFE.

News and Notes

FOREIGN APPOINTMENTS.—Dr. Weygandt has been appointed Adjunct Professor of Psychiatry at the University of Würzburg.

Privatdocent Dr. Hans Gudden has been appointed Adjunct Professor of Psychiatry at Munich.

Privatdocent Dr. Heinrich von Holben has been appointed Professor of Psychiatry at the University of Lembeck.

Dr. J. B. Janssens has been appointed Assistant in Psychiatry at the University of Leyden.

Dr. H. Jaeger has been appointed Assistant in Psychiatry at Gröningen.

Dr. T. W. Polet has been appointed Assistant in Psychiatry at the University of Utrecht.

Dr. Robert Gaupp has been appointed Director of the Psychiatric Clinic at Munich.

Dr. Paul Schroeder, Assistant in the Psychiatric Clinic at Breslau, has been made Privatdocent.

Dr. Johannes Finckh has been made Assistant to the Psychiatric Clinic at Tubingen.

The International Congress of Psychiatry, Neurology and Hospital Treatment for the Insane will meet in September, 1907, in Amsterdam.

NERVOUS DISEASES RESEARCH FUND.—The first annual meeting of the subscribers to the Nervous Diseases Research Fund was held on April 4th at the National Hospital for the Paralyzed and Epileptic, Mr. Edgar Speyer in the chair. The report for 1904 stated that 48 autopsies had been performed, and that the results of the investigations would prove useful in diagnosis, and in solving some serious problems of cerebral and spinal disease. Among the cases of tumor of the brain there were six in which the brain stem was affected, and the clinical and pathological examination had added to the known signs of disease in that region, and had drawn attention to the functions of certain cells and nerve fibers in the mid-brain. Information of a similar character had been obtained in reference to tumors of the cerebellum. This would be of great value in the diagnosis of the exact situation of such tumors or of other morbid conditions affecting the same part. Special attention had been given to the study of myasthenia gravis, a disease which was almost invariably fatal and about which little was known. New facts concerning its pathology had been ascertained and these facts, such as they were, afforded a hope that the condition might be curable by medical treatment. The results of the work were embodied in papers which would be available for the medical profession generally. The contributions to the fund in 1904 amounted to £731, and there was a balance at the banker's at the end of the year of £459 18s. 4d. Mr. Edgar Speyer was elected Chairman and Treasurer for the current year, and Mr. J. Danvers Power, Honorary Secretary. Dr. M. Gordon Holmes was appointed director of the research work of the fund.

THE AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION.—DR. BURR CHOSEN PRESIDENT.—The American Medico-Psychological Association, in session at San Antonio, Tex., recently elected Dr. C. B. Burr, of Flint, Mich., president for the ensuing year. The action of the association carries with it a merited recognition of the eminence which Dr. Burr has attained in the medical profession, and a practical expression of his services in behalf of the association as secretary and treasurer for seven years, and as vice-president of the organization during the past year.

Originally organized in 1844 as an association of medical superintendents of American institutions for the insane, the American Medico-Psychological Association, the oldest national medical organization in America, was reorganized under its present name in 1892, when its membership was enlarged to include eminent alienists, neurologists and others especially interested in the care of the insane. The present membership of the association is about 425, and includes prominent alienists in both the United States and Canada.

Dr. Burr joined the association in 1890, the year following his election to the superintendency of the Eastern Michigan Asylum for the Insane, at Pontiac, Mich. He had previously been assistant physician and assistant medical superintendent of the asylum since 1878. In 1894 he resigned the superintendency of the Eastern Michigan Asylum to become medical director of Oak Grove. He served as secretary and treasurer of the American Medico-Psychological Association from 1897 until 1904, when he was elected vice-president.,

Dr. Burr had planned to attend the convention at San Antonio, but was prevented from making the trip by an injury which he sustained by being thrown under his horse as a result of the animal stumbling and falling with him while he was out riding nearly three weeks ago.

CRAIG COLONY APPOINTMENT.—Dr. L. Pierce Clark has been appointed consulting Neurologist at the Craig Colony for Epileptics, Sonyea, N. Y.

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VOL 32 JULY, 1905 No. 7

THE

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Nervous and Mental Disease

Original Articles

PRESIDENTIAL ADDRESS.

THE IMPORTANCE IN CLINICAL DIAGNOSIS OF PARALYSIS OF ASSOCIATED MOVEMENTS OF THE EYEBALLS (BLICKLAEHMUNG), ESPECIALLY OF UPWARD AND DOWNWARD ASSOCIATED MOVEMENTS.[1]

BY WILLIAM G. SPILLER, M.D.,

PROFESSOR OF NEUROPATHOLOGY AND ASSOCIATE PROFESSOR OF NEUROLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

To preside over this distinguished body of American neurologists is an honor that your chairman feels too deeply to express adequately in words.

> "* * * like a cipher,
> Yet standing in rich place, I multiply
> With one 'We thank you' many thousands more,
> That go before it."

Our membership list now is almost full, and we shall be obliged to raise the number above one hundred or to reject desirable applicants. There is some danger in doing the former. When there are many vacancies a candidate is more likely to be accepted without careful scrutiny of his record than when admission is of necessity limited. On the other hand it would be unfortunate to bar out desirable men. A moderate increase in our membership may be advantageous, and the Council will suggest the placing of the limit at 110.

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[1] Read at the meeting of the American Neurological Association, June 1, 1905. From the Neuropathological Laboratory of the University of Pennsylvania.

It is proposed to make some important changes in the Congress of American Physicians and Surgeons. The subject has already come before this Association but must be considered again. The following resolution is offered: Resolved: That in the opinion of this Society it is desirable that the Executive Committee of the Congress of American Physicians and Surgeons of 1907 should consider the advisability of altering the constitution of the Congress, and report progress thereon to the several Societies interested at their sessions of 1905 and 1906.

In preparing this address it has seemed best to me to follow a custom that on occasions such as this has been more common abroad than at home, and to discuss a subject of general interest. I therefore present for your consideration the results of some investigations that may in a small degree increase our knowledge of the disorders of the nervous system.

Disturbances in the associated movements of the eyes (Blicklähmung) afford a sign of localizing value in lesions of the brain, and yet this subject has received comparatively little attention. V. Kornilow, who has written the best recent paper on these palsies, remarks that clinical cases carefully studied are rare, and even more so are those with necropsy.

The opinion of Parinaud and Sauvineau that upward or downward associated ocular paralysis is always associated with paralysis of convergence is incorrect, as shown by a case reported by Parinaud himself, by a case reported by v. Kornilow, and by other cases. It is true, however, as Parinaud pointed out in 1883, that in paralysis of associated ocular movements diplopia often is absent. Indeed when in such palsy diplopia later appears it may be a sign of improvement, as it was in one of the cases of my series (Case 5), where the disappearance of the palsy in one eye caused diplopia, soon followed by complete recovery. Although paralysis of associated ocular movement of any form is usually a sign of grave significance, one should be careful about giving a fatal prognosis. In a case reported by Raymond and Cestan[1] the paralysis lasted ten years, and in a number of cases complete recovery has occurred (Case 5 of my series, and other cases), while in others improvement has been very marked (Case 8 and Case 9, of my series).

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[1] Raymond and Cestan. Revue Neurologique, 1901, p. 70.

Having had the opportunity recently, through the kindness of many colleagues, to whom credit is given in the detailed reports, to study four cases (Beier,[2] Cases 1, 2 and 3), in which paralysis of lateral associated ocular movements occurred, and nine cases in which paralysis of upward or downward associated ocular movements was a striking sign (including three of the four cases mentioned above); having also examined microscopically the material obtained from four of these cases, I have selected paralysis of associated ocular movements as the subject of my address. I shall not devote much space to the study of the paralyses of lateral associated movements, as these have received much attention, and I[3] have recently discussed this subject. The evidence is strong that this form of paralysis is indicative of a lesion of the posterior longitudinal bundle near the sixth nucleus. The external rectus muscle on the side of the lesion may be more affected than the internal rectus of the other eye, and this according to Parinaud[4] is the rule. He mentions also that the movement of convergence may be greater in the eye in which the external rectus is paralyzed, because of the paralysis of the external rectus.

The disturbances of the upward or downward associated movements have been studied less than those of lateral associated movement. The former, though usually acquired, may be congenital, and Oppenheim[5] has observed congenital paralysis of associated upward movement. Uhthoff[6] speaks of paresis of upward associated movement with slight impairment of movement in other directions, occurring in multiple sclerosis.

We have reason to believe that the cerebral cortex exerts some control over the associated movements of the eyeballs; especially strong is the evidence concerning lateral conjugate movements. Conjugate deviation of the eyes has been studied within the past few years by Klaas,[7] and from his investigations of the literature, it appears that this phenomenon was first observed by Andral (1834). It was studied by Duplay, Durand-Fardel and others, but Prevost showed that conjugate deviation of the eyes often occurs in hemiplegia and has nothing to do with strabismus, as Andral believed; he showed further that it is often associated with deviation of the head; that the deviation is toward the lesion when the lesion is cerebral, and away from the lesion when it is in the isthmus. Landouzy believed that irritation and paralysis produce deviation in opposite directions when the lesion is in the cerebrum, and Grasset showed that the patient looks at his paralyzed limb when the lesion is a paralyzing one in the brain stem, and at the lesion when it is an irritating one, and in this part of the brain. Wernicke assumed that the rapid disappearance of conjugate deviation of the head and eyes is owing to the partial control of the lateral ocular movements by the cerebral hemisphere of the same side, and to the action of this hemisphere in place of the injured opposite cerebral hemisphere. Wernicke believed that conjugate deviation of the eyes is caused by a lesion of the lower parietal lobe, and he reported three cases of lesion in the lower part of each parietal lobe in which voluntary lateral associated ocular movements were lost, pseudo-ophthalmoplegia, although in another case with similar lesions disturbance of the ocular movements was not observed. The evidence concerning a center for lateral movements of the eyes in the lower parietal region or angular gyrus is conflicting. There is more evidence that a center for lateral associated ocular movements is situated in the posterior part of the frontal lobe.

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[3] Potts and Spiller. Univ. of Penna. Med. Bul., Dec., 1903.

[4] Parinaud. Archives de Neurologie, Vol. 5, No. 14, 1883, p. 145.

[5] Oppenheim. Lehrbuch der Nervenkrankheiten, 4th Ed., Vol. 2, p. 706.

[6] Uhthoff. Archiv. für Psychiatrie, Vol. 21, 1890, p. 379.

[7] W. Klaas. "Ueber konjugierte Augenablenkung bei Gehirnkrankheiten," Inaugural Dissertation, Marburg, 1898.

The evidence concerning a center for lateral movements of the eyes in the lower parietal region or angular gyrus is conflicting. There is more evidence that a center for lateral associated ocular movements is situated in the posterior part of the frontal lobe.

The recent investigations of Grünbaum and Sherrington by electrical irritation have again demonstrated that a center for ocular movements probably exists in the frontal lobe. Pathological alteration of the frontal lobe has given evidence to the same effect. In a case of cerebral syphilis with severe lesion of the right frontal lobe, reported by Karl Schaffer,[8] paralysis of associated movement of the eyeballs to the left occurred, and Schaffer concludes from a study of this case that a center for lateral associated movements exists in the posterior part of the second frontal convolution, although he does not deny that there may be another and similar center in the supramarginal and angular gyri.

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[8] Schaffer. Neurogisches Centralblatt, Nov. 16, 1904, p. 1035.

Hemianopsia cannot be regarded as the cause of conjugate deviation in all cases, although much has been attributed to it recently by certain French writers. Dejerine and Roussy[9] have demonstrated that it is not necessarily the cause of the deviation, inasmuch as deviation existed in a patient of theirs who had been born blind and whose cortical visual zone had not been developed.

So far the knowledge gained relates chiefly to associated lateral movements, but we know much less concerning a cortical center for upward or downward associated movements.

The views of Parinaud and some other writers on this subject lack confirmation. They believed that there is a center for ocular movements in the occipital lobe and a more important one in the frontal lobe. The inferior part of the latter center controls the upward movement of the eyes, the superior part controls the downward movement of the eyes, the intervening part controls the lateral movements. It is true that lateral associated movements have been impaired with paralysis of upward or downward associated movements in a number of instances, as they were in some of my cases, but a lesion in the vicinity of the corpora quadrigemina will better explain this form of paralysis than a lesion of the cerebral cortex. Teillais says regarding his own case, it would be unreasonable to regard the lesion as cortical, because if the lower part of the cortical center controls the upward movements, and the upper part the downward movements, then the whole of this center except the middle portion must have been destroyed, as the lateral movements were preserved. Such a view cannot be accepted.

Paralysis of lateral associated movement from cortical lesions, as seen in the conjugate deviation of the eyes, is always, so far as I know, transitory, and the persistence of such paralysis points to a lesion of the posterior longitudinal bundle. We have little or no anatomical or pathological evidence that is really valuable concerning the existence of a cortical center for upward or downward associated movements, but in reasoning from analogy we must assume that such a center or centers exist. The clinical case that Parinaud[10] reported in 1892 as indicating a lesion of the frontal center, is in the light of our present knowledge unsatisfactory because of the association of homonymous lateral hemianopsia.

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[9] Dejerine and Roussy. Revue Neurologique, Feb. 15, 1905, p. 161.

Parinaud reported the case before the Society of Ophthalmology, April 5, 1892. The man had paralysis of the lower left part of the face, of the left half of the tongue, and temporarily of the left hand. He had almost complete paralysis of the elevators of the eyeballs, with a little reduction in the extent of the lateral movements. Convergence was well performed. (Here Parinaud presents a case of paralysis of upward associated movement with preservation of convergence.) Downward movement was normal. Parinaud believed there was a lesion in the right frontal lobe, at the level of the lower part of the Rolandic fissure, involving the centers for the face, tongue, hand, and associated movements of the eyeballs.

This patient had transitory partial left homonymous hemianopsia, and this was supposed to be the result of a lesion near the visual zone, which occupies, according to the teaching of Parinaud in 1892, all the occipital lobe, and extends to near the ascending parietal convolution. This opinion we now can hardly accept.

Hysteria may cause paralysis of the associated ocular movements, but there is no pathognomonic sign of this form of the palsy, and in some instances a correct diagnosis may be extremely difficult or even impossible. No case presents this difficulty more clearly than the one reported by Crouzon, Marie and Babinski[11] before the Neurological Society of Paris on three different occasions in 1900 and 1901.

Crouzon presented the case as one of spasmodic tic, later Babinski and Parinaud expressed the opinion that it was organic and caused by a supranuclear lesion, and still later Marie presented the case with the diagnosis of a purely functional disorder, a neurosis, as a spasm of the elevators of the eyeballs. When the patient threw his head as far backward as possible and followed with his eyes a finger lowered slowly the eyeballs moved downward, but if his head were erect and he attempted on command to look at his feet, he bent his head forward and the eyeballs went forcibly upward. After thirty or forty seconds the spasm disappeared and the eyeballs returned to the normal position. Marie from this concluded that there must be a spasm of the elevators and not a paralysis of the depressors. The disorder had occurred after an apoplectic attack lasting seventeen hours, and the man had become blind. During the attack he had not had stertorous breathing nor passage of urine or feces, and this Marie regarded as remarkable if there were an organic lesion. The coma disappeared suddenly, but the patient was delirious for several weeks and did not recognize anyone or his surroundings. This Marie regarded as like hysteria. The speech was slow and hesitating, and the visual fields were contracted.

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[10] Parinaud. Annales d'Oculistique, Vol. 107, p. 283, 1892.
[11] Revue Neurologique, 1901, p. 428.

Parinaud pointed out that the downward movement in following a finger is a reflex act and better performed than a voluntary movement, and this he says he has observed in hysteria, but when the condition is hysterical there is no inconvenience from the ocular disturbance, just as there is no inconvenience from the contraction of the visual fields in hysteria, and Parinaud has never seen a hysterical patient incline the head forward in order to make use of the superior part of the *champ du regard* (Dr. de Schweinitz suggests as a suitable translation for this term, field of fixation). The dissociation between the voluntary and reflex movements is not always hysterical. Parinaud has pointed out that spasm may occur in sound ocular muscles as the result of paralysis of other ocular muscles when the patient is required to look in different directions. A paralysis confined to one eye may be the cause of spasm in the other, so that the sound muscles may appear to be affected. In these cases the paralysis is peripheral, but even in associated palsies it is not rare to see disturbance of the antagonistic muscles. In conjugate lateral paralysis from lesion of one sixth nucleus there is almost always nystagmus when the patient looks toward the sound side, and this nystagmus may be so pronounced that it may be difficult to say which side is affected. This I also have noticed in a case of complete paralysis of lateral associated movements on one side and of partial paralysis on the other. In the case which called forth so much discussion in the Neurological Society of Paris the visual fields were contracted only in the inferior portion and not concentrically, therefore the contraction was not like that of hysteria. The *champ visual* (visual field) Parinaud distinguishes from the *champ du regard* (field of fixation). He believes that if the cortical center for associated movements of the eyes in the frontal lobe be destroyed, the voluntary associated movements will be lost, but the reflex associated movements will be preserved, and yet little, if any, clinical evidence in support of this opinion can be found.

Babinski pointed out that in the case under discussion the downward movement of the eyes was imperfect with the head thrown back. He quoted the case reported by Schröder[12] in which only paralysis of the associated downward movement persisted, and the eyes could not be lowered below the horizontal plane. Any attempt to look downward caused a spasm of the elevators and eyes turned upward.

Oppenheim[13] has observed cases of pseudobulbar paralysis in which lateral movements of the eyeballs were impaired in voluntary innervation, but were preserved when the patient tried to follow an object or to turn toward the direction from which a sound came.

All the pathological evidence that I have been able to obtain in cases of persistent palsy of associated upward or downward movement is indicative of a lesion near the aqueduct of Sylvius. It is extremely doubtful whether a lesion confined to the corpora quadrigemina and causing no pressure on the surrounding parts ever causes paralysis of associated ocular movements, and those who favor such a view have not produced proof of a supranuclear center in this part. Bernheimer[14] says Topolanski showed that electrical irritation of the corpora quadrigemina of rabbits does not produce ocular movements, and that these parts could be removed without producing symptoms. He believed that there is a center for ocular movements near the oculomotor nucleus.

Bernheimer, from experiments on monkeys, has shown that the ocular movements do not depend on the integrity of the corpora quadrigemina, that this part of the brain does not contain a reflex center for these movements, and that after destruction of this part the ocular movements are normal. He obtained synergic ocular movements from irritation of the angular gyrus both before and after he had removed the corpora quadrigemina. He thus refutes Prus' statements that a center for ocular movements exists in the corpora quadrigemina.

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[12] Schröder, quoted by Teillais, Bul. de la Soc. Fran. d'Ophthal., 1899, p. 415. (See my abstract of this case.)

[13] Oppenheim. Neurologisches Centralblatt, 1895, p. 40.

[14] Bernheimer. Wiener klin. Wochenschrift, No. 52, 1899, p. 1310.

Cases have been reported in which the corpora quadrigemina have been destroyed without disturbance in the movements of the eyeballs (Weinland, Seidel, Ruel, Nissen, cited by v. Kornilow).

In Case 2 of my series there was complete paralysis of right associated lateral movements, and great impairment of left and of upward associated movements. The paralysis of lateral associated movements is to be explained by the involvement of both posterior longitudinal bundles. If we assume that the nuclei of the superior rectus and inferior oblique muscles are in the posterior part of the oculo-motor nucleus, we can understand why marked impairment of upward movement was present in this case, but this is contrary to the views held by some concerning the position of these groups of nerve cells.

Siemerling[15] has recently remarked that the numerous experimental studies of Bach, Bernheimer, van Gehuchten and von Biervliet have not led to uniform conclusions. The oculomotor nucleus is merely the place for the transference of impulses and we are not in a position to state which parts are concerned with the innervation of the individual muscles.

My Case 1 is further evidence that the nuclei of the superior rectus and inferior oblique muscles may be in the posterior part of the oculomotor nucleus, because there was paresis of upward associated movement and the nerve cells of the oculomotor nuclei were not diseased, but the posterior part of the nuclei was affected by pressure from the tumor in the pons, and the aqueduct of Sylvius was compressed upon the left side. The tumor did not invade the nuclei of the third and fourth nerves. The paralysis of left associated lateral movement is explained by the almost complete destruction of the left half of the pons by a tubercle, and thereby involvement of the posterior longitudinal bundle. The left sixth nerve was also much degenerated. In both Cases 1 and 2 downward movement was normal, this would seem to be evidence that the nucleus of the inferior rectus muscle must be in advance of those for the superior rectus and inferior oblique muscles, as in each case the tumor grew from the pons and did not invade the oculomotor nucleus.

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[15] Siemerling. Archiv. für Psychiatrie, Vol. 40, No. 1, p. 61.

If this is the correct position of these nuclei, a lesion developing ventrally in the pons as shown in two of my cases may by pressure upon the posterior part of the oculomotor nuclei cause paralysis of associated upward movement, as a result of injury of the nuclei for the superior rectus and inferior oblique muscles, and it would not be at all necessary to assume the existence of a coordinating center for upward associated movement. Paralysis of downward associated movement depends on impairment of the inferior rectus and superior oblique muscles, these having cells of origin in two distinct nuclei. There must be a close association between the nucleus of the superior oblique and that of the inferior rectus muscle of the same side, and possibly of the opposite side. It is probable therefore that a lesion of the nuclei of the inferior rectus muscles and of the fibers connecting them with the nuclei of the trochlear nerves would cause paralysis of associated downward movement, and it seems unnecessary to assume that a coordinating supranuclear center for upward or downward movements exist in the corpora quadrigemnia or their vicinity. In deed two of my cases (Cases 1 and 2) show that such a center is improbable, because in each case the lesion causing paresis of upward associated movement was posterior to the oculomotor nucleus, and did not implicate the corpora quadrigemina. Three of the cases of my series afford further evidence against a center for coordinate ocular movements in the corpora quadrigemina. In one the paralysis at first (Case 4) was only in upward movements, later also in lateral associated and in downward associated ocular movements. In this case therefore a paralysis at first confined to one form of associated movement later extended to other forms. In another case (Case 5) the paralysis of upward associated ocular movements disappeared, leaving only paresis of the left superior rectus, and finally this also disappeared. In Case 9 the associated palsy disappeared, leaving paralysis of muscles only on one side. It is very common to have a palsy of one or more branches of the oculo motor nerve in association with paralysis of associated upward or downward movement, and this is more easily explained by a lesion of the oculomotor nucleus than by a cortical lesion. Cortical centers control associated movements, not isolated muscles.

The paralysis of the sixth nerve occurring with paralysis of associated upward or downward movements has been attributed by Lichtheim to pressure upon the nerve at the base of the brain. The opinion held by Parinaud, although supported by Duval, Laborde and Graux, can hardly be accepted, viz., that the sixth nucleus gives nerve fibers to the internal rectus muscle, and that this muscle has a double innervation, the sixth nerve for lateral movement, the third for convergence.

The assumption of a basal coordinating center above the oculomotor nucleus for ocular movements seems forced. The lateral associated ocular movements can be well explained by the connection formed by the posterior longitudinal bundle between the nucleus of the branch to the internal rectus muscle and the nucleus of the sixth nerve. It is reasonable to suppose that certain fibers connecting the different parts of the oculomotor nucleus with one another, or connecting the group of nerve cells innervating the inferior rectus with the nucleus of the trochlear nerve have a coordinating function similar to that of the posterior longitudinal bundle, and that this function may be disturbed by a small lesion. It is striking that the paralysis of downward associated movement (necessitating the implication of two separate nuclei of ocular muscles) without paralysis of upward associated movement is exceedingly rare, far more so than is the isolated paralysis of upward associated movement.

Conjugate deviation of the eyeballs from lesions of the pons is not common when the lesion is confined to one side of the pons. In a case of hemorrhage into the tegmentum of the pons (see Case 3) that I have had the opportunity to study and the brain from which I have examined and refer to in this paper, inward deviation of only the right eye occurred from paralysis of the sixth nerve. One gets the impression that conjugate deviation of the eyeballs away from the side of the lesion is common in lesions of one side of the pons, but in four cases of lesions of this part observed by me (three of tumor and one of hemorrhage) causing paralysis of the external rectus muscle of the same side, I have looked for the conjugate deviation but failed to find it. It is probable that in most cases the side of the pons opposite to the lesion is directly or indirectly involved. . . . . .

Von Monakow[16] believes that when the lesion implicates the sixth nucleus of one side, apart from the paralysis no disturbance, i.e., no deviation occurs, unless it is the result of secondary contracture. I believe that conjugate deviation is rare even as a late sign of paralysis of one sixth nucleus. Of course any involvement of both sixth nuclei would make conjugate deviation impossible, and in one of my cases (Case 2) both these nuclei were affected.

In a case of softening of the dorsal part of the left side of the pons reported by Albert Ransohoff[17] there was complete paralysis of the associated ocular movement to the left and of convergence movement of the right eye. The right external rectus muscle was not over-active, but the left internal muscle was in marked spasm. The left abducens nucleus was involved in the lesion, the right escaped, but the right posterior longitudinal bundle was involved. Ransohoff mentions that Wernicke and Jolly have shown that paralysis of the external rectus muscle does not in itself cause contraction of its antagonist, the internal rectus, and that in the cases of these authors, as well as in his own, there was a lesion of the opposite posterior longitudinal bundle. In Wernicke's case a tumor had destroyed the left abducens nucleus and invaded the right side of the pons. Both eyeballs were persistently turned to the right, especially the left eyeball.

According to the study of the literature by Ransohoff, contracture of the unparalyzed antagonistic muscles, but not always in both eyes, and more in the internal rectus muscle on the side of the lesion than in the external rectus muscle on the side opposite to the lesion, was observed in the cases of Wernicke, Jolly, Graux, Meyer (the lesion did not extend to the mid-line),

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[16] Gehirnpathologique, p. 355.

[17] Ransohoff, Archiv. für Psychiatrie, Vol. 35, 1901, p. 416.

Spitzer (slight, and after one-half year duration of the lesion), Mierzejewsky and Rosenbach, Gebhard, and Ransohoff.

Ransohoff says that if the posterior longitudinal bundle, as is assumed by most authors, contains a part of the voluntary fibers for the internal rectus muscle of the opposite side, a lesion of these fibers, as of the pyramidal fibers, must cause spastic contracture of the muscle they innervate. Therefore, according to Ransohoff, contracture of the internal rectus muscle on the side of the lesion indicates that the posterior longitudinal bundle of the opposite side is also involved, but this contracture probably could not exist if the nucleus of each abducens were destroyed.

In four of my cases (Cases 1, 2, 3, and Beier) both posterior longitudinal bundles must have been much affected and conjugate deviation did not occur in any.

In one case (Case 4) in which paralysis of lateral movements was present, the movement of either eyeball to the right or left was distinctly greater when one eye was covered than in associated lateral movments. The individual movements of the eyeballs were therefore greater than the associated movements.

One of the cases (Case 9) was especially interesting in that syphilitic infection was positive. The paralysis of associated movement was peculiar; the downward movement was completely lost in the right eye and much impaired in the left; the upward movement was slight in the right eye and completely lost in the left; the lateral associated movements were nearly normal. Almost all the ocular movements were regained except the downward movement of the right eye. It is remarkable that the complete paralysis in the associated movements was in the downward direction in one eye and in the upward direction in the other, although the associated movement was very feeble. It is remarkable that the associated palsy disappeared leaving paralysis of downward movement of the right eye.

In four cases of my series with necropsy (Beier, Cases 1, 2, and 3) the pupil was smaller on the same side as the lesion of the pons. This alteration of the size of the pupil from a lesion of the pons is difficult to explain, but the presence of the sign in four cases shows that it is of diagnostic significance, and also that the fibers controlling the iris pass through the pons.

In at least two cases nystagmus was present in looking away from the side of the chief lesion (Beier, Case 2). This was caused by weakness of the opposite external rectus muscle from pressure upon or direct involvement of the nucleus of the abducent nerve. This sign may be expected when a lesion affects the dorsal part of the pons and destroys the function of one abducent nucleus and weakens that of the other. Nystagmus may surely be the result of paresis of ocular muscles.

I have studied 38 cases of paralysis of upward or downward associated movement reported in the literature, not including my own 9. In many cases certain symptoms are not referred to, so that statements regarding the frequency of symptoms are incomplete. V. Kornilow used only 20 cases of the 27 to which he found references in drawing his conclusions. As a result of my study of these 47 cases.

Paralysis of upward associated movement without paralysis or paresis of downward associated movement was found in 26 cases (Henoch, Priestley Smith (2), Nieden, Gowers, Parinaud (2), Ormerod, Reinhold, Thomsen, Lichtheim (3), Hoesslin, Bruner, Mills, Noguès and Sirol, Kornilow, Posey, Parkinson, cases 1, 2, 4, 5, 6, 8, of my series).

Paralysis of upward associated movement with paralysis or paresis of downward associated movement was found in 16 cases (Wernicke, Parinaud, Hope, Nothnagel, Eisenlohr, Verrey, Sharkey, Sauvineau, Teillais, Gordinier, Raymond and Cestan (slight), Kornilow, Cases 3, 4, 7, 9, of my series).

Paralysis of upward associated movement with impairment of lateral movement, often developing later, was found in 15 cases (Henoch, Priestley Smith (2), Lichtheim (3), Parinaud, Poulard, Raymond and Cestan, Kornilow, Parkinson, Cases 1, 2, 3 and 4 of my series).

Paralysis of upward associated movement without impairment of lateral movement was found in 22 cases (Wernicke, Nieden, Gowers, Parinaud (2), Ormerod, Hope, Thomsen, Sharkey, Sauvineau, Hoesslin, Bruner, Teillais, Gordinier, Noguès and Sirol, Kornilow, Posey, Cases 5, 6, 7, 8, 9 of my series).

Paralysis of downward associated movement without paralysis of upward associated movement was found in 5 cases

(Gee, Poulard, Curzon-Marie and Babinski, Basevi, Schroeder), but in none of my nine cases.

The reaction of the iris was found to be impaired in 14 cases (Henoch, Gowers, Parinaud, Thomsen, Eisenlohr, Lichtheim, Sharkey, Sauvineau, Hoesslin, Gordinier, Mills, Kornilow, Posey, Case 9 of my series) and is said to have been normal in 4 cases (Parinaud, Teillais, Poulard, Raymond and Cestan), but in many cases no statements regarding the iritic reflex are made, and in all my nine cases the iritic reflex was preserved.

The optic nerve was found to be affected in 15 cases (Gowers, Thomsen, Nothnagel, Gee, Eisenlohr, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Cases 1, 5, 7, 9 of my series), and normal in 12 cases (Wernicke, Nieden, Parinaud (2), Ormerod, Reinhold, Sauvineau, Teillais, Raymond and Cestan, Mills, Noguès and Sirol, Posey).

Convergence was impaired in 15 cases (Parinaud (2), Sharkey, Sauvineau, Teillais, Poulard, Gordinier, Raymond and Cestan, Kornilow, Basevi, Cases 2, 4, 5, 8, 9 of my series), and normal, or nearly normal, in 9 cases (Hope, Parinaud, Noguès and Sirol, Kornilow, Posey, Cases 1, 3, 6, 7 of my series.

Ptosis was found in 7 cases (Reinhold, Eisenlohr, Gordinier, Kornilow, Nothnagel, Cases 2 and 8 of my series), and was said to be absent in 13 cases (Wernicke, Parinaud, Gee, Sauvineau, Teillais, Raymond and Cestan, Cases 1, 3, 4, 5, 6, 7, 9 of my series).

Necropsy was obtained in 19 cases (Henoch, Wernicke, Gowers, Hope, Reinhold, Thomsen, Gee, Eisenlohr, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Nothnagle, Basevi, cases 1, 2 and 3 of my series.)

In all of these cases except one (Thomsen[18]) parts about the aqueduct of Sylvius were implicated, and in Thomsen's case a gumma was found in the cerebral peduncles. Thomsen cannot explain why the intense degeneration of the right oculomotor nerve and the slight degeneration of the left oculomotor nerve caused paralysis of only upward movement. It is possible that the Nissl method, if it had been employed, would have shown marked alteration of the oculomotor nuclei, possibly as a toxic effect from the adjacent gumma, and in this way the paralysis of upward associated movement may have been caused. Such was the condition in one of my cases (Case 2), unless perhaps the alteration of the nerve cells was caused by pressure. Until at least another case similar to Thomsen's is reported we may well hesitate to believe that a peripheral lesion of one oculomotor nerve may cause paralysis of only upward associated movements.

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[18] Thomsen. . Archiv. für Psychiatrie, Vol. XVIII., 1887, p. 616.

Tumor was found in 14 cases (Henoch, Gowers, Hope, Reinhold, Gee, Lichtheim (3), Sharkey, Hoesslin, Gordinier, Nothnagel, cases 1 and 2 of my series), a bullet wound in one case (Eisenlohr), apoplectic cyst in one case (Wernicke), hemorrhage in one case (my Case 3), and lesion uncertain in one case (Basevi).

Recovery or partial recovery occurred in 7 cases (Priestley Smith, Nieden, Verrey, Kornilow, Cases 5, 8 and 9 of my series). Important symptoms other than ocular palsies were found in 41 cases (Henoch, Wernicke, Priestley Smith (2), Gowers, Parinaud (3), Hope, Reinhold, Thomsen, Nothnagel, Gee, Eisenlohr, Lichtheim (3), Verrey, Sharkey, Sauvineau, Hoesslin, Bruner, Teillais, Poulard, Gordinier, Çruzon-Marie and Babinski, Raymond and Cestan, Mills, Nogués and Sirol, Kornilow (2), Schroeder, Posey, Parkinson, Cases 1, 2, 3, 4, 6, 8 and 9 of my series).

As a result of my studies I believe that persisting paralysis of associated lateral movement indicates a lesion of the posterior longitudinal bundle; that persistent paralysis of associated upward or downward movement indicates a lesion in the vicinity of the oculomotor nucleus, and that paralysis of associated ocular movements is not the result of a lesion of extracerebral nerve fibers. Lesions of the cerebral cortex may certainly cause paralysis of lateral associated ocular movements, and possibly of upward or downward associated ocular movements, but cortical paralysis of associated ocular movements is transitory, unless possibly where the center on each side of the brain is destroyed. Paralysis of associated ocular movements may be caused by hysteria. Any case in which associated ocular palsy is persistent and is of organic nature, is unsuitable for operation unless the operation is merely palliative, as the lesion is probably within the posterior part of the pons or cerebral peduncle, according to the form of the associated palsy, or else causes much pressure upon the dorsal portions of these structures. The paralysis of associated ocular muscles may be produced by inflammatory lesions or lesions of a similar character (alcohol, syphilis) as well as by tumor, and may disappear later in the course of the disease. Syphilitic ependymitis and cellular infiltration must always be considered in diagnosticating the lesion causing paralysis of associated ocular movements.

CASES OF PARALYSIS OF ASSOCIATED UPWARD OR DOWNWARD MOVEMENT OBSERVED BY DR. SPILLER.

Case 1. Tubercle of left side of the pons.

Galik, was admitted to my wards in the Philadelphia General Hospital, Aug. 11, 1904, and died Oct. 22, 1904.

He said he had been paralyzed for nine months on the right side of the body. He was a fairly well developed man, aged thirty-one years. He was examined Aug. 12 by Dr. T. H. Weisenburg, when the following notes were made:

He never had headache nor vertigo, and has had no difficulty in chewing or swallowing, this statement being corroborated by the nurse. There is no dribbling of saliva. He has internal deviation of both eyes, more of the left. The right pupil is larger than normal, the left is normal in size. Both pupils respond to light and in accommodation and convergence. The left external rectus is completely paralyzed, and the right external rectus is partially paralyzed. All of the other movements are well performed. On showing his teeth he can draw back the right angle of his mouth better than the left. The right nasolabial fold is more distinct than the left. On attempting to close his eyes the left lids remain partially separated. He wrinkles the right side of the forehead normally, but there is hardly any movement on the left side. The tongue deviates to the right when protruded, but is not atrophied and shows no tremor. The masseter muscle does not contract so well on the left side as on the right.

He is able to lift the right upper limb from the bed for a few minutes, but the movement is unsteady and weak. Resistance to passive movement at the right elbow is almost lost. The right upper limb seems somewhat spastic. The right forearm is held in supination, the hand flexed on the wrist, and the fingers flexed into the palm. The grip is very feeble. There are no contractures, but the fingers are stiff. The whole right upper limb is wasted, more so in the distal parts, the muscles of the thenar and hypothenar eminences being almost completely atrophied. Atrophy of the extensors and flexors of the forearm is more marked on the right side than it is in the corresponding muscles of the left upper limb. The biceps, triceps and wrist reflexes on the right side are prompter than normal.

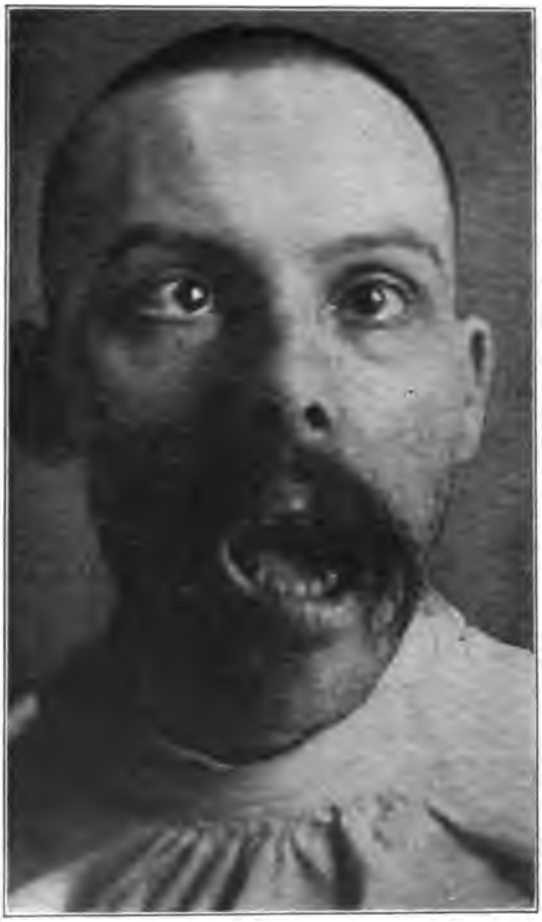

Fig. 1. Internal deviation of both eyes, especially of the left, and deviation of the lower jaw to the left from paralysis of the left muscles of mastication. Paresis of associated movements upward and to the left.

Sensation for touch is felt in the right upper limb, right side of chest and abdomen, but less so than in the left side. He does not recognize a pin prick over these parts on the right side as a pin prick, but as touch, and sensation is more impaired in the distal parts. The left upper limb is normal in every respect. He is not able to recognize objects placed in his right hand.

The power of the right lower limb is diminished. He is able to flex and extend the thigh, but is not able to move the ankle joint or the toes. The motor power in the left lower limb is normal. The patellar and Achilles tendon reflexes are much increased on the right side, but not on the left. Sensations for touch and pain are diminished in the right lower limb, and more so in the distal parts; they are normal in the left lower limb. The right lower limb is somewhat spastic and atrophied, and the atrophy is seen especially in the thigh muscles. He has had no bladder or rectal disturbances.

He says he has no pain anywhere.

His speech is thick and indistinct.

Dr. Van Epps saw the patient Aug. 13, and made the following notes: Contraction of the left orbicularis palpebrarum is markedly weakened, that of right is questionably weakened. Right pupil is round, 3 mm. in size. Left pupil is round, 2 mm. in size. Both pupils react to light and in accommodation and convergence. Outward rotation of O. D. 2 mm. short; internal rotation 3 mm. short, and upward 2 mm. short of full; downward rotation normal.

O. S. inward rotation normal, complete absence of outward rotation, upward and downward rotation similar to that of fellow. On this date some weakness of the right orbicularis palpebrarum was noticed. The nurse said the patient had difficulty in swallowing.

Dr. Van Epps made a further report Aug. 16, 1904.

O. D. Under homatropine, pupil 6 mm. round; media clear, marked optic neuritis with swelling of retina for 1½, disk clean in all directions (neuroretinitis). Disk elevation 2 diopters. Arteries reduced in size, veins slightly increased in caliber and quite tortuous, partly disappearing here and there in swollen nerve and retina. Hemorrhage on upper part of disk. Middle and peripheral zones normal. O. S. Under homatropine 4½ mm., round. Cornea epithelium so eroded that a view of the fundus is impossible.

Entire loss of corneal and conjunctional reflex on the left side (hemianesthesia of same complete).

There seems to be marked hyperesthesia of eyelids and surrounding cutaneous area on the left side, but patient's answers are contradictory.

He was seen by me Aug. 25, at which time I made the following notes:

In converging the movement of the right eyeball inward is about equal to that of the left, and is greater than when the attempt is made to look toward the left with both eyeballs. He has paralysis of associated movements, both in looking to the left and upward. The movement of each eyeball upward is impaired equally. When he closes his eyelids he has marked fibrillary tremor of the left upper lid. He has marked fibrillary tremors of the left masseter and the left masseter is completely paralyzed. When the mouth is opened the jaw goes markedly toward the left. Speech is distinctly bulbar. Hearing cannot be tested carefully on account of slow cerebration, but seems to be affected on the left side. The weakness of the right upper and lower limbs is very pronounced, though it is not complete paralysis. The tendon reflexes of the right upper and lower limbs are distinctly exaggerated.

He is able to stand, but not without support.

He swallows with difficulty.

The left cornea is anesthetic.

He says pin prick is felt more in the left side of the face. He draws up the corner of the mouth when the corresponding side of the face is stuck with a pin. This sensory condition was noticed also by Dr. Weisenburg, who found (Aug. 12, 1904) that he recognized touch and pain sensations distinctly better on the left side of the face than on the right.

Sept. 21. Speech is very indistinct and is bulbar in character. The left side of the face is paralyzed. Marked fibrillary tremors are seen in both sides of the tongue, and the tongue deviates to the right when protruded. The left pupil does not reach the median line, and the right pupil goes a little beyond the median line in trying to look to the left. Pain and tactile sensations are diminished in the right upper limb. Babinski's sign is absent on each side. Ankle clonus is not present on either side.

Sept. 23. He does not hear a low ticking watch when placed on the left ear, but does hear it when it is placed on the right ear. He has excessive appetite.

Sept. 28. The left eye is much congested, although mosquito netting is kept constantly over his face to keep the flies away and his eyes are washed frequently with boric acid. Pus is found in the anterior chamber of the eye.

Oct. 22. He has been gradually sinking more than ten days. Respirations have become shallow and rapid, and he is unable to speak. He died at 8.30 p.m., Oct. 22, 1904.

Death occurred while the patient was in the service of Dr. C. K. Mills, after I had gone off duty. I am indebted to Dr. Mills for the pathological material.

At the necropsy ulcerative tuberculosis of the lungs and a large tumor in the left side of the pons were found.

Sections made through the cerebral peduncles show that the aqueduct of Sylvius is compressed on the left side. The left cerebral peduncle is considerably large than the right. The tumor extends into the lower part of the left cerebral peduncle. The left fourth nucleus appears to be almost normal, a few of the cells are slightly altered, but the nucleus on the whole is remarkably well preserved. The posterior longitudinal bundle on the left side is pushed more dorsally than is the corresponding bundle on the right side. The right 4th nucleus also is normal except that a few of the cells show alteration. Most of the nerve cells in the oculomotor nucleus, both in each lateral and in the median group seem to be normal, only occasionally a cell can be found in which the chromophilic elements have undergone disintegration.

Left 2nd nerve. There is a slight round cell infiltration of the pia and of the septa extending into the optic nerve; the nerve does not appear to be degenerated in its intracranial portion.

Left 3d nerve appears to be entirely normal.

Left 6th nerve is much degenerated. The medullary sheaths are swollen. The axis cylinders are also swollen and in some places have disappeared. One side of the nerve is much more altered than the other. The Weigert stain shows much degeneration at one side of the nerve where the medullary sheaths stain very badly. The right sixth nerve appears to be normal.

Left 7th nerve shows a moderate swelling of medullary sheaths.

Left 8th nerve appears to be normal.

Left 5th nerve is almost completely degenerated in a few bundles that probably belong to the motor part of the nerve.

The left pyramid is much degenerated by the method of Marchi, as is also the spinal root of the left fifth nerve. There are some degenerated fibers on each side of the medulla oblongata posterior to the lower olive, more on the left side, and also in the left lemniscus.

The right crossed pyramidal tract is much degenerated by the Marchi method from the cervical into the lumbar region. The left direct pyramidal tract is much degenerated in the cervical region.

There is no meningitis about the medulla oblongata or spinal cord. The nerve cells of the anterior horns of the cervical and lumbar regions stain well by the thionin method.

Weigert hematoxylin sections show intense degeneration of the left anterior pyramid, of the left direct pyramidal tract, and of the right crossed pyramidal tract.

The tumor in the middle portion of the pons, antero-posteriorly, invades a little the right side of the pons (see Fig. 2), but the greater part of the right pyramidal tract is not involved in the growth. The aqueduct of Sylvius is much compressed and appears as a long narrow slit. Almost the entire left half of the pons is destroyed, a narrow zone at the ventral part escaping. The tumor in transverse section is nearly round. Microscopically the tumor is not sharply defined from the surrounding tissue, and its peripheral zones consist of mononuclear cells with round nuclei. The central portion of the tumor is degenerated, and the cells do not stain well. The tubercle bacillus was not found. The tumor has the appearance of a tubercle.

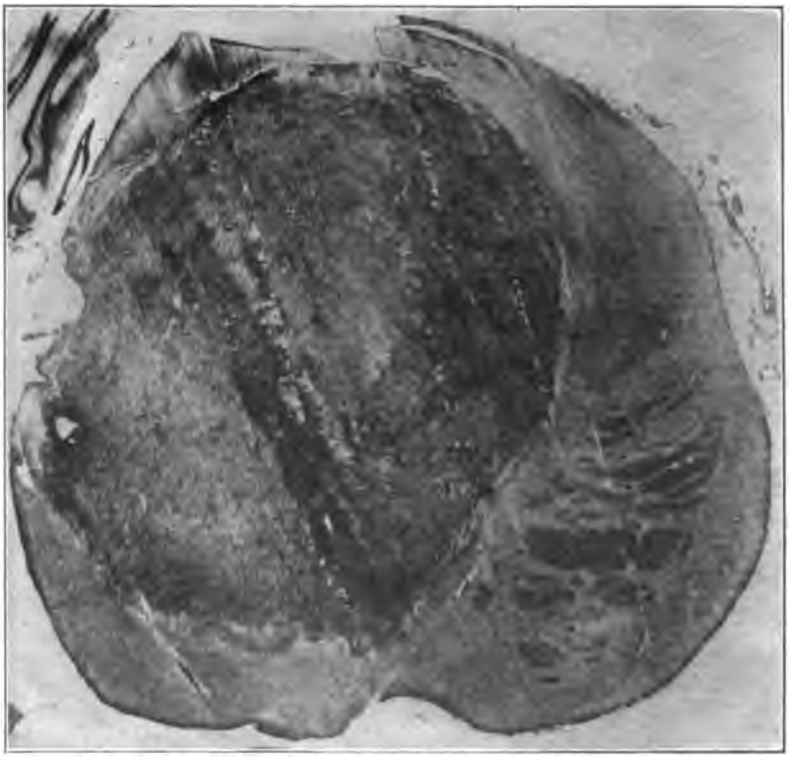

FIG. 2. Tubercle in the left side of the pons.

Summary: B. Galik, a male aged thirty-one years, had been paretic (Aug. 11, 1904) nine months in the right limbs. He was paretic in both sides of his face, especially in the left side, and in the left muscles of mastication, and had fibrillary tremors of the left masseter muscle, and of the right side of the tongue. The tendon reflexes in the right upper and lower limbs were exaggerated. Diminution of sensation and some wasting were observed in the right upper and lower limbs. The movements of both eyeballs upward were impaired, and there was complete loss of movement of the left eyeball to the left, and marked impairment of movement of right eyeball to the left; also, impairment of movement of right eyeball, but not of left eyeball, to the right. Downward movement of both eyeballs was normal, and convergence was preserved (paresis of associated movements upward and to the left). He had bulbar speech, impaired hearing in the left ear, anesthesia of the left cornea, optic neuritis, at least in the right eye (left disc not visible because of corneal opacities) ; and later left neuroparalytic keratitis.

The necropsy showed a large tubercle involving almost the entire left side of the pons and invading a little the right half of the pons and extending into the lower part of the left cerebral peduncle. Most of the nerve cells of the oculomotor nuclei appeared to be normal, but those of the left nucleus were displaced. The left 3d nerve was normal. The left sixth nerve and spinal root of the left 5th nerve were much degenerated, and the left 7th and left motor 5th nerves were partially degenerated. The left pyramid was degenerated. The aqueduct of Sylvius was much compressed from the left side.

The slight voluntary movements of the right upper and lower limbs may be explained by the preservation of a narrow zone in the ventral portion of the left half of the pons and by axis cylinders that probably passed through the tubercle without being destroyed. It has been shown that axis cylinders may be found within a tubercle even after the medullary sheaths have been destroyed.

The paralysis of associated left lateral movement is to be explained by the implication of the left posterior longitudinal bundle. The paresis of upward movement could not have been caused by implication of the corpora quadrigemina, because this portion of the brain stem was not involved. The nerve cells of the oculomotor nuclei were slightly degenerated, but the left oculomotor nucleus was much displaced in its posterior portion, and the function of the nerve cells here must have been interfered with.

The pupil in this case was smaller on the side of the tumor.

It seems difficult to explain the absence of paralysis of associated ocular movement to the right: unless it is because the ocular examination was made two months before death occurred, and at that time the turbercle had not extended to the right side of the pons. The man's intelligence became considerably affected before death.

Case 2. Glioma of the right side of the pons.

A. Moore, male, aged sixteen years, was a patient of Dr. Wm. Evans, from whom the following notes were obtained:

His parents and two sisters were living and well. His maternal grandfather died of apoplexy at seventy-one years. He had always had good health until the early part of July, 1904, when he began to feel nervous, slight exertion made him tired, he became dizzy, had headache and a tendency to turn to the left, and he staggered slightly in walking. His sight became dim, and at times he had diplopia.

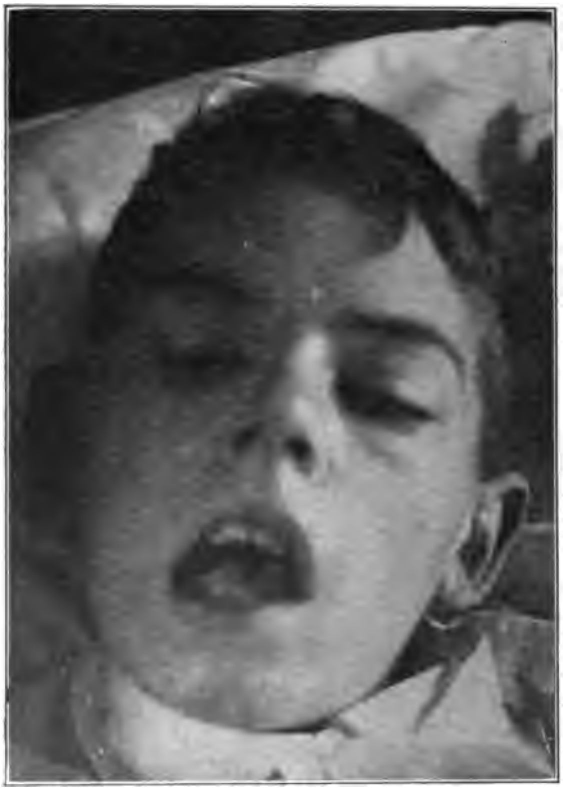

Fig. 3. Attempt to converge. Notice position of left eye. The left eye is moved more toward the right than when the attempt is made to move both eyeballs to the right (see Fig. 5). Notice the marked deviation of the lower jaw to the right when the mouth is opened, this being the result of paralysis of the right external pterygoid muscle.

He was seen by Dr. C. S. Potts Aug. 15, 1904, and notes made by him at that time are as follows:

He sits with the head retracted and bent to the left, the chin pointing to the right, although he can hold it straight if he makes an effort to do so. He has ptosis of the left eyelid. The pupils are equal and respond to light and in accommodation. Some weakness of the external rectus of the right side is detected and he says that he sees double when he looks to the right. Coarse nystagmic movements are present when the eyes are moved laterally in either direction. The angle of

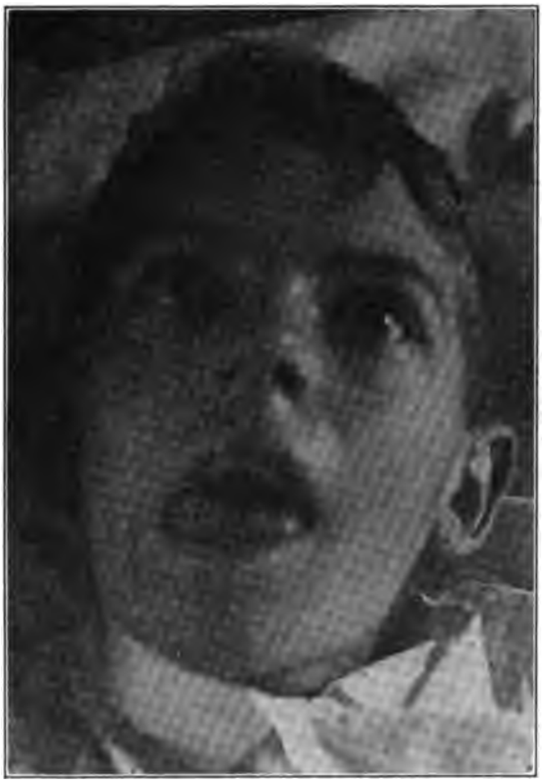

FIG. 4. Attempt to look upward and to protrude the tongue. The eyeballs move very slightly upward. The lower jaw goes to the right, the tongue to the left when protruded. The dot on tongue shows the point of the tongue. On great effort, the tongue could be protruded a little further, and the deviation to the left was greater.

the mouth droops slightly on the right side, but there is no distinct paralysis. The tongue seems to protrude slightly to the left. The gait is staggering, but there is no tendency to fall to one side more than the other. Resistance to passive movements is good. The patellar reflexes are exaggerated, the left more than the right, and he has slight ankle clonus on the right side and marked clonus on the left side. Plantar reflexes are diminished, the toes move in neither direction on the right side, but slightly upward on the left side. The arm jerks (biceps, triceps, wrist) are apparently equal on both sides and about normal in degree. He hears the tick of a watch in the right ear at 12 inches, in the left ear at 3 feet. Bone conduction is better on the left side. He can recognize the direction of passive movements of the fingers and toes, but cannot touch the end of his nose with either index finger with certainty. He has not other sensory symptoms except headache.

An examination of the eyes by Dr. Krall, Aug. 17, 1904, gave the following results:

He has partial ptosis of left eye, paralysis of the left external rectus, or rather a paresis, paresis of the left inferior rectus, and a nystagmus to the right on attempting fixation. Accommodation for his age is good. The pupils are nearly alike on the average (the left one is ½ mm. larger than the right), they react normally to light, accommodation and convergence. His vision is practically normal. The ophthalmoscopic examination shows no involvement of the optic nerve. Field of vision is, however, contracted concentrically for form and color. Color perception is good. No color blindness.

The patient was seen by me Oct. 11, 1904, in consultation with Dr. Evans, and at that time I made the following notes:

The left pupil is considerably larger than the right, Iritic reflex is prompt in each eye. Nystagmoid movement in trying to look to the left. Movement of eyeballs to the left imperfect. Left iris does not reach the external canthus and the eyeball returns almost immediately towards the median line. Unable to look towards the right with either eye, pupils remaining in the median line when he attempts to look to the right. Able to converge very slightly with the left eye, and does so with the right. Upward movement of each eye is almost lost, the pupils hardly passing beyond a line drawn horizontally across the centers of the palpebral fissures. Downward movement seems almost normal. He closes the eyelids firmly on each side. Slight drooping of each lid is present. He wrinkles the forehead well and equally on both sides. The movements of the face are feeble on each side in the lower distribution of the seventh nerve. He does not show the teeth well nor does he draw up either corner of the mouth well. Tongue when protruded goes distinctly towards the left. Left masseter muscle contracts, the right apparently does not. When the mouth is opened the lower jaw goes far to the right. The right orbicularis palpebrarum is distinctly weaker than the left, and often in closing his lids, the right lids remain partially open. Pin prick is promptly perceived on each side of the face. It is probable that the right side of the face in the lower distribution of the facial nerve is a little weaker than the left side. The neck is not stiff. There is a tendency for the head to turn to the left, but it can be turned easily to the right. He hears a low-ticking watch 6 to 8 inches in the left ear. Cannot hear it in the right ear when placed against the ear. Speech is thick, bulbar in type, not at all aphasic. He counts fingers correctly

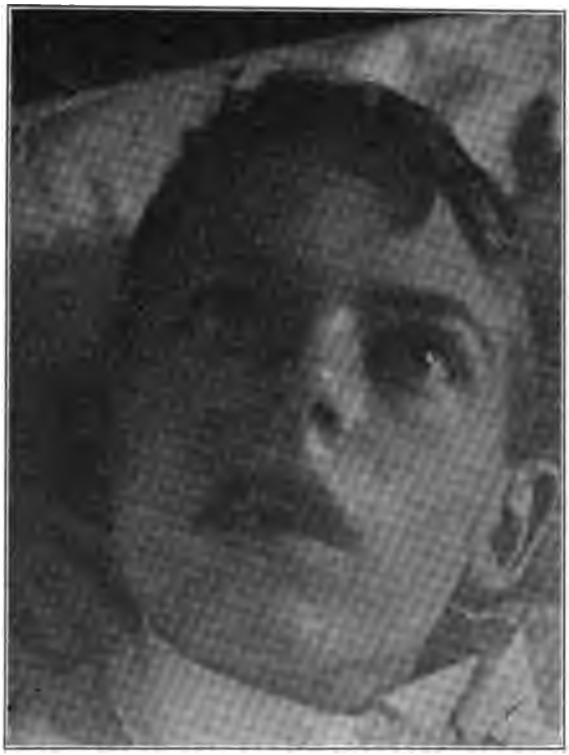

FIG. 5. Attempt to look to the right and to separate the lips with the teeth together, showing weakness in the lower distribution of the facial nerve on each side. Notice the position of the left eye in this photograph and in Fig. 3, showing attempted convergence.

held before either eye. He is able to swallow liquids or semisolids without much trouble. The soft palate moves freely on voluntary effort. Finger-to-nose test shows extreme ataxia with right hand. The left upper limb is almost completely paralyzed, but there is slight movement of the left shoulder. No motor power is preserved below the shoulder in the left upper limb. Biceps and triceps reflexes are slightly increased on the left side, almost normal on the right side. Sensations for touch and pain are somewhat diminished in the left upper limb, and are normal in the right upper limb. Sense of position seems to be preserved in the left upper limb, as he puts his right hand directly on his left with eyes closed, when told to do so. Upper

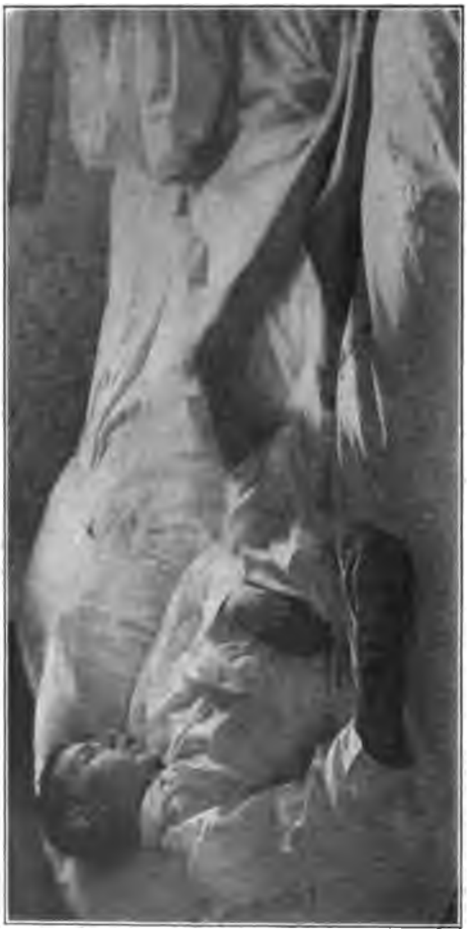

Fig. 6. Showing the tendency of the head to fall forward and to the left; also the complete paralysis of the left upper limb and the partial voluntary movement of the left lower limb at the hip. The voluntary power at the left shoulder was very slight.

limbs are not distinctly wasted. Left lower limb is distinctly weaker than the right, but not as weak as the left upper limb. The right lower limb has normal power. The lower limbs are not distinctly wasted although the whole body is somewhat emaciated. The patellar reflex is slightly increased on the right side, distinctly so on the left. Tendency to patellar clonus is present on the left side only. Persistent ankle clonus on both sides, but much more distinct on the left side. Typical Babinski sign is obtained on the left side, but not on the right side, there being no movement of the toes at all on this side. Sensations for touch and pain are probably diminished slightly in left lower limb. Heel to knee test shows much ataxia of right lower limb. When sitting on the edge of the bed he would fall if not supported, and head falls far forward, and to the left. When made to stand he would fall if not supported. He cannot stand erect, and ataxia is so great that he does not seem to know where to put his legs. His knees flex under him. He understands all that is said to him. Sitting up makes him dizzy. He has ocasional attacks of dull headache in the right temporal region. The tongue is not wasted, nor does it show fibrillary tremor. Almost every day he has attacks of nausea, sometimes with vomiting.

In converging left eyeball moves distinctly a little more toward the right than when he tries to look to the right with both eyeballs.

Further notes were made by me Oct. 23, 1904.

Upward movements of eyeballs much impaired, but seem to be better than when he was last seen. The jaw goes far to the right. Tongue not protruded much beyond the lips, but deviation to the left occurs. Movement of eyeballs to the right is lost, much impaired toward left and nystagmus occurs when eyes are turned toward the left. Weakness of right orbicularis palpebrarum is more pronounced. Right eyeball moves up a little more than does the left. He raises the left eyebrow better than the right. He does not hear a loud-ticking watch unless placed directly on the right ear, but hears it a foot away from the left ear. Sensations for touch and pain are not much impaired on either side of the face. He certainly feels touch and pain in the left forearm. The movement in the left lower limb is almost entirely at the hip. He feels both touch and pain sensation in the left lower limb. He has no movement in the left toes or at the left ankle. When he sits up the head falls far forward and to the left and the whole body inclines forward.

The necropsy was obtained Nov. 10, 1904.

The left 4th nucleus is partly degenerated, some of the nerve cells of this nucleus show much degeneration, but most of them are in excellent condition. There is no distinct group of cells constituting the right 4th nucleus at this level, because the right 4th nucleus has been pushed more cerebralwards by the tumor. The right posterior longitudinal bundle has been pushed dorsally toward the aqueduct of Sylvius, and the aqueduct is distorted in such a way that its right half is pushed further dorsally than its left half. The glioma extends into the right cerebral peduncle as high as the level of the fourth nucleus, but does not invade this nucleus. Some of the cells of the right fourth nucleus are degenerated, but most of them are normal.

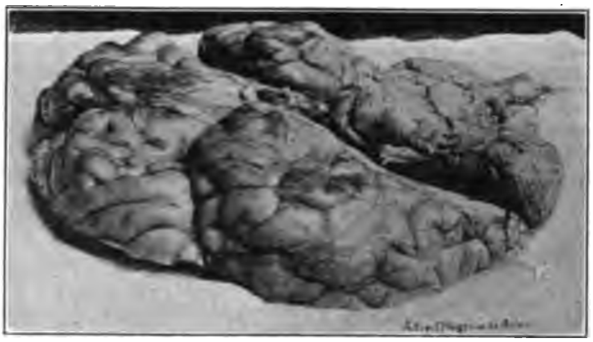

FIG. 7. Glioma of the right half of the pons, invading the lower left half of the pons.

The median group of cells in the oculomotor nucleus is intensely altered, the chromophilic elements have become fine granules and the nuclei are displaced far to the periphery of the nerve cells. The left lateral group in the oculomotor nucleus is well developed, although here and there a nerve cell is found that is not quite normal. The right lateral group shows much alteration, and yet not nearly so much as the median group. The nerve cells are much less numerous in the right lateral group than in the left lateral group, and they are pushed backward toward the aqueduct of Sylvius; many of them appear to be normal. Cells bordering on the upper and median side of the right lateral group are intensely degenerated. The right oculomotor nerve in its extrapeduncular portion does not appear to be degenerated.

The right second, fifth and sixth nerves and the left sixth nerve do not appear to be degenerated either by the Weigert hematoxylin or acid fuchsine method.

The right half of the medulla oblongata is much larger than the left, but does not contain any tumor tissue at the level of the twelfth nuclei. The twelfth nuclei and the intramedullary part of the twelfth nerves appear to be normal. The right anterior pyramid takes the Weigert hematoxylin stain a little less deeply than the left, but the degeneration is slight.

Sections from the medulla oblongata stained by the Marchi method show some recent degeneration of the right anterior pyramid, and of each column of Burdach, but not of the columns of Goll.

Sections from the uppermost part of the cervical cord show considerable recent degeneration of the left crossed pyramidal tract, and of the columns of Burdach, and slight recent degeneration of the right crossed pyramidal tract.

The comparatively slight degeneration of the pyramidal tract from the right side of the brain is striking in consideration of the size of the tumor.

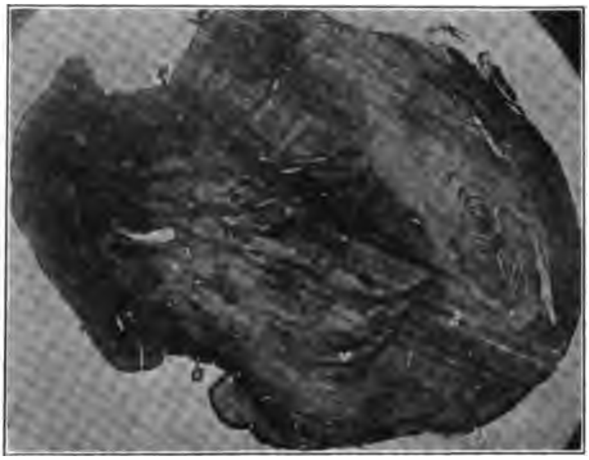

FIG. 8. Microscopical section through the upper part of the pons, showing the glioma of the right half of the pons. FG=raphe of pons.

A section made through the upper part of the pons shows that the tumor is entirely in the right half of the pons, does not reach the raphe, and leaves intact many bundles of the pyramidal tract on the right side, although they are more widely separated from one another than is normal. The right lemniscus is not degenerated. The tumor is chiefly in the lateral part of the right side of the pons, and here is cystic. The tumor consists of cells containing small round or slightly oval nuclei and are separated from one another by much intercellular fibrous tissue. The tumor is a glioma, and in some places is very vascular. It invades the left side of the pons nearer the medulla oblongata.

Summary: A. Moore, male, aged fifteen years, (Oct. 11, 1904) had been complaining about nine weeks. The left pupil was larger than the right. Iritic reflexes prompt. The movements of the eyeballs to the left were imperfect, and to the right lost. Upward movement was much impaired, downward movement about normal. Convergence preserved, but impaired. (Paralysis of upward and lateral associated movements.) Slight bilateral ptosis. Weakness of each facial nerve and of the left side of the tongue. Paralysis of the right masseter muscle and of the left limbs. Tendency to fall to the left. Impairment of hearing in the right ear. Bulbar speech. Diminution of sensation in the left limbs.

A large glioma was found in the right half of the pons, extending into the right cerebral peduncle and the right side of the medulla oblongata, and in the lower part of the pons involving the left side of the tegmentum of the pons. The nerve cells of the 4th and 3d nuclei were partly degenerated, especially on the right side. The left crossed pyramidal tract showed considerable recent degeneration.

The slight impairment of sensation may be explained by the slight involvement of the lemniscus, but it is remarkable that the left motor paralysis was so intense when so much of the motor tract in the right side of the pons escaped. In the left upper limb the only voluntary movement was slight drawing up of the shoulder, and in the left lower limb slight flexion at hip and knee.

(*To be continued*)

DISPENSARY WORK IN NERVOUS DISEASES.[1]

By Smith Ely Jelliffe, M.D., Ph.D.,

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From the first day of January, 1904, to December 31 of the same year, 1,981 new patients were entered on the books of the Department of Nervous Diseases in the Vanderbilt Clinic. Some 2,138 patients applied as suffering from nervous disorders, but of these only the number mentioned were considered suitable for examination.

The total number of patients in the entire clinic may be briefly stated as follows:

DEPARTMENTS	NEW PATIENTS
Nervous	2,138
Orthopedic	975
Surgical	4,470
Medical	13,358
Children	3,430
Gynecological	2,684
Eye	4,326
Ear	1,519
Nose and Throat	4,117
Skin	3,642
Genito-Urinary	2,356
Total	42,995

From a brief glance it may be seen that the general incidence of nervous diseases to the clinical population is about as it has been in years past, approximating five per cent.

The total number of visits last year in the nervous department, 9,722, was less than the year previous. The extremely severe late winter of 1903-1904, and the early and equally severe winter of 1904-1905 may in part account for this, as many of the patients suffering from organic spinal affections have found it extremely trying to attend the clinic regularly.

The personnel of the clinic remained much the same as during the year 1904.

Dr. Pierce Bailey, Chief of Clinic; Chas. E. Atwood, M.D.; C. M. Haviland. M.D.; B. E. Krystall, M.D.; L. P. Clark, M.D.; S. E. Jelliffe, M.D.· L. S. Manson, M.D.; S. P. Goodhart, M.D.,

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[1] Report of Clinic of Prof. M. Allen Starr for the year 1904.

H. R. Humphries, M.D.; L. M. Gibson, M.D.; Thomas P. Prout, M.D.; E. L. Hunt, M.D.; G. W. Todd, M.D.

After examination of the 1,981 patients it was found that no organic or functional disorder of the nervous system could be found in 46 women and 94 men, while for 24 women and 24 men the diagnosis of the disease could not be determined with sufficient accuracy to be entered as a diagnosis on the history records.

This leaves a total of 1,790 patients concerning whom statistical information is herewith presented. Of these 879 were male and 911 female.

Mental Disease.—As in previous reports for 1902 and 1903 the statistics show that mental troubles represented about ten per cent of the cases coming under observation, the women preponderating, 137 compared with 78 men.

Mental defectives were 68 in number, 3.8 per cent; distributed as follows: Idiocy 9, boys 1, girls 8; Imbecility, 59, boys 8, girls 51.

The disparity in the number of imbeciles of the sexes is to be interpreted as purely accidental, although Mayo-Smith says in his *Statistics and Sociology* that in England there is an excess of males. He includes the figures of adult idiots of all classes, whereas in this clinic children only are concerned.

With reference to the Insanities the stand taken last year in our report may again be stated. It is believed that a thoroughly good diagnosis of many of the insanities cannot be made by a single examination at a clinic of the general type of the Vanderbilt Clinic particularly since in many instances dependance must be placed entirely upon the history offered by the patients themselves. Thus the diagnosis must be accepted in the most general sense, a convenient classification based upon the ascertainable and frequently defective history.

Of the adolescent insanities Dementia Præcox was diagnosed in 12, 6 men and 6 women; they were for the most part examples of the purely hebephrenic grouping. Katatonic rigidity was present in one of the patients.

Simple hypochondriasis or depression of sufficiently deep grade to merit the classification Melancholia was found in 31 patients, 15 men and 16 women.

Manic-depressive insanity was diagnosed in four instances, 2 men, 2 women. In all of these regular cycles had occurred. Secondary and senile dementias were present in five patients.

General Paresis was diagnosed in 26 patients. Four of these were women. The total number is the same as for last year but there were no women on the list for 1903. There were 434 patients suffering from this disease admitted to the New York State Hospitals during the year, or 7.7 per cent of the entire number of the insane admissions in the hospitals for the insane of the State.

Paranoid states, allied more closely to Paranoia itself than to other syndrome groupings were present in 6, 4 men and 2 women.

Minor Psychoneuroses were not uncommon. We have preferred to group these borderland cases thus: Morbid fears, 3; Menstrual Psychoses of mild grade, 32; Occupation Psychoneuroses, 11; Sexual Perversions, 1; and Neurotic, unmanageable children with bad dreams, 4.

NERVOUS DISEASES.—Sensory-motor Neuroses represent the greater number of affections in the clinic population. Thus in 1904 there were 326 patients suffering from Neurasthenia and 77 from Hysteria.

Of the neurasthenics, so-called, 194 were men and 132 women. Seventy-five per cent of the men were Russian Hebrews. The type of neurasthenics here represented are for the most part purely mental cases. Laziness, indifference, weakness of mind, and supersensitiveness characterize them all. They are for the most part ill because of lack of moral courage, and being temporarily out of work, the great majority come to get some medicine and are rarely heard of a second time. They are a very unsatisfactory kind of patient to have to treat.

Hysteria, or the hysterical temperament, was diagnosed in 77 patients, 8 males and 69 females. Only a few of these women presented the more severe grades of the affection. We cannot help but feel that major hysteria is a comparatively rare affection with our native population. The major hysterias seen are almost invariably in patients of foreign birth or the descendants of recent arrivals. It might be said, however, that the particular type of clientele of the Vanderbilt Clinic is drawn from a social stratum in which hystericals are less apt to be numerous.

The Motor Neuroses were represented as follows: Epilepsy, 173, 90 males and 83 females; no unusual features were brought out in the histories of the epileptics seen in 1904.

The general feeling that Sydenham's Chorea has been more than usually prevalent during the year is confirmed by this year's study. There were 221 patients, 85 males and 136 females this year, a total incidence of 12 per cent of the entire nervous population. Assuming the dispensary-seeking population of the State of New York for 1904 to be 1,000,000—it was 932,587 in 1900, according to the reports of the State Commissioners of Charities, and assuming a similar proportion to exist in other dispensaries in the State, there would be 6,000 children who suffered from chorea in New York State during 1904. The great majority of the children suffering from chorea are between the ages of 5 and 14 years. In New York State there were, according to the census of 1900, 1,357,510 children between the ages of 5 and 14 years. Thus, judged by crude statistical inquiry, one child in about every 200 of the population suffers from this disease.

One patient with what was thought to be Friedreich's Paramyclonus Multiplex was admitted for treatment, a boy of about 10 years of age. Habit spasms were present in seven girls; spasmodic Wry Neck in 2; miscellaneous forms of Tic in 6, and Stammering in 28—10 males and 18 females.

Paralysis Agitans occurred in 16, 10 men and 6 women. Nothing unusual was encountered in the histories of these patients.

LESIONS OF PERIPHERAL NERVES.—The total number of sufferers from peripheral nerve injuries of one type or another was 323, males 180 and females 143.

Neuralgia was dignosed as follows:

	MEN	WOMEN
Generalized and non-localized	4	15
Occipital	1	1
Supraorbital	4	11
Trigeminal	10	18
Facial, including dental	9	10
Brachial	1	3
Intercostal	—	2
Sciatic	24	5

Following the clinic usage of separating the neuralgias from the neuritides, the following figures were obtained:

	MALE	FEMALE
Neuritis, not localized by observer	12	7
Alcoholic neuritis	13	—
Alcohol and pressure (Saturday night neuritis)	6	1
Auditory	1	—
Lead neuritis	8	—
Mercurial neuritis	1	—
Typhoid (post-infectious neuritis)	—	1
Diphtheritic (post-infectious neuritis)	1	2
Malarial (post-infectious neuritis)	—	3
Measles (post-infectious neuritis)	—	2
Rheumatic (post-infectious neuritis)	3	1
Traumatic	6	6
Optic	1	—

Peripheral palsies were present in almost double the number seen in 1903, 123 in all, 68 males and 55 females. The distribution was as follows: Facial paralysis (Bell's palsy) 46 men, 23 women. Exposure is the almost universal history. In a few, trauma has been the cause, and in others, middle ear disease.

Paralysis of the ocular muscles was observed in one man, and paralysis of the vocal cords in one man and in one woman.

Involvement of the musculo-spiral, next to the facial, is observed in the greater number of patients, 21 men and 4 women showing typical pressure symptoms. Erb's palsy was diagnosed in 14 children. Brachial and nerve palsy was present in 12 men and

7 women. Other isolated paralyses were: Circumflex, 4 men; Ulnar, 2 men; Median, 1 man; Anterior tibial, 1 male, 1 female; Serratus, 1 male, 1 female.

SPINAL CORD AFFECTIONS.—The Anterior Poliomyelitis syndrome was observed in 33 patients. Of these 28 were acute and 5 chronic in character. Typical Anterior Poliomyelitis of children occurred in 19 boys and 9 girls. The chronic types were in adults, 4 men and 1 woman. No instance of high anterior poliomyelitis, bulbar or other involvement was seen during the year.

Diffuse Myelitis was observed in one male only.

Multiple Sclerosis was observed in 9 patients, 4 women and 5 men. The diagnosis is uncertain in two of the cases, a cord tumor being suspected in one.

The Posterior Scleroses have been observed in 24 instances. Tabes Dorsalis occurred in 23 of these, 18 men and 5 women. Defective development of the posterior columns, Friedreich's Ataxia in 1 girl. The comparative ratio of men to women suffering from tabes this year is high.

Lateral Sclerosis was diagnosed in 6 men; Spinal Tumor in 1 woman; Syringomyelia in 3 men and 2 women; Hemorrhage into the cord in 2 men. Two instances of Fracture Paraplegia were recorded, and one of Paraplegia due to Potts' disease, all in men.

BRAIN—Organic affections of the brain were present in 111 patients.

The meninges were involved in 3, 1 man, 2 women. The types of lesion were thought to be Chronic Pachymeningitis.

Apoplexy was diagnosed in 51 patients, 38 men and 13 women. No attempt has been made to differentiate between hemorrhage, thrombosis and embolism. The vast majority of the hemiplegias, however, were hemorrhagic in origin. Infantile cerebral hemiplegia was diagnosed in 16 instances, 3 boys and 13 girls. Infantile diplegia was present in 3 girls.

Brain syphilis was suspected in 5 men and 1 woman, 6 in all. Two men suffered from brain tubercle. Two cerebral tumors and 2 cerebellar tumors were diagnosed. One patient with atrophy of the cerebellum was admitted. Cerebral arteriosclerosis was thought to be present in 24 patients, 17 males and 7 females. Concussion of the brain was dignosed in 7 patients, 6 of whom were men.

TROPHONEUROSES.—Exophthalmic goitre, 4 men and 12 women, 16 in all; Myxedema, 1 man; Meralgia Paresthetica, 1 man; Erythromelalgia, 1 woman; Raynaud's disease, 1 woman.

MISCELLANY.—Alcoholism, 46 men, 1 woman; Nicotinism, 2 men; Morphinism, 1 man; Headache, 18 men, 49 women; Chronic Hiccough, 1; Deaf Mutism, 2 females; Sleeplessness, 3 men, 7 women; Narcolepsy, 1 man; Pseudomuscular Hypertrophy, 1 female; Acroparesthesia, 5 men, 12 women; Sunstroke, 1; Joint Atrophy, 1; Tremor, 1; Coccyodinia, 1: Blepharospasm, 1.

DELUSIONS OF THE INSANE.[1]

By Robert H. Chase, M.D.,
OF PHILADELPHIA.

For some months past the hospital staff of Friends' Asylum has been giving special attention to the different aspects of delusions of the insane and at the same time, incidentally, to a variety of kindred types of morbid mentality. In looking about for suitable material to meet the present purpose, it seemed probable that a brief presentation of the main conclusions reached in the study would not be without interest to the members of this society.

It may be stated at the outset that the distinctive feature of our thesis is the assumption that delusions of the insane are based primarily upon a change in the vital feelings, and not, as commonly asserted, on disturbances solely in the intellectual activities of the mind. Lest this bare statement be not sufficient to make its meaning clear to you, let us review briefly the relative influence of the feelings upon ideas, as well as the influence of ideas upon the feelings.

The principle is well established that vital feelings, the psychical corollary of organic sensations, accompany all of our ideas in the stream of consciousness. But it should be remarked also that the feeling-element in a mental state is not subject to the law of the association of ideas like our thoughts. Here may be seen the influence of ideation upon feeling, for it is entirely through this relation of thoughts to new thoughts that feelings pass into new feelings. Of the two, thought is by far the more mobile. Hence it is that feelings form, as it were, the basis to which the results of experience are only gradually transferred from the more fleeting surface of thought. Since feeling is so deeply rooted in consciousness, a profound conviction or belief requires time as a necessary condition of its development. Hence those ideas which have taken root in the feelings are not easily displaced. Owing to this quality the feelings have a retarding effect upon the combining of ideas in the train of thought. This may be termed the inertia of the feelings, which not only explains some of the peculiarities of morbid thinking, but also becomes the source of many inconsistencies in daily life.

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[1] Read at a meeting of the Philadelphia Neurological Society, March 28, 1905.

In normal conditions knowledge gains in strength and security, and becomes truly a personal possession only when rooted in the feelings in this way. In the same manner it can be seen how a delusion becomes deeply entrenched in the insane mind. Furthermore, the fact that a certain idea, or set of ideas, has as a basis strong interest or deep emotion, alters its relation to other ideas. It becomes a stronger center of association than it otherwise would. In all experiences attention is given only to that which in some way affects one's deepest interest. Feeling simply becomes the dictator. All ideas that are not in accord with the ruling feeling are either cast aside or suppressed. Even ideas which stand in connection with oft-repeated experience may be wholly ignored, where there is this strong tension or deep and enduring interest. The Christian Scientist, as an example, lays more stress on the few instances in which he can believe that he has received help by his peculiar process of healing, than on the many in which such a belief is impossible. The reasoning of the medical hobbyist is open often to the same criticism. This is the explanation of the failure of many vaunted new remedies. Again, when love is all absorbing the unlovable traits of the object are not seen. Some one has said, "Love is blind, only because it is wonderfully keen sighted in one certain direction." A widely divergent set of facts seen in criminal annals equally illustrates this truth. In the murderer, the passion that prompts him to the execution of the deed often overpowers all rational prudence, causing a reckless disregard of incriminating details. Lotze has especially emphasized the fusing of ideas with the given vital feeling. If the vital feeling is changed, the road to the ideas connected with it is blocked. It is in this way that he interprets the facts that after recovery from severe illness, one does not remember what he experienced while it lasted, or that one cannot recall the particulars of a terrifying dream.

In consequence of the inertia of feeling. the accompanying feeling does not at once change with the idea, but extends to the succeeding ideas, even when these are in no way connected with that which produced the feeling. Thus, a feeling is often strange to ourselves, but in most cases it exerts its influence upon the new content of thought without our noticing it. All strong feeling, therefore, struggles for the sole control in the mind and gives color to all mental activities. The final questions, with which the views of life are concerned, are decided in the last resort according to the dictates of feeling. The attitude is the same whether the man be sane or insane. This is no less clearly shown in the great contrast of the views of the optimist and the pessimist in society, than in the cases of the expansive paretic and of the gloomy melancholic of the asylum. Just as the appearance of a landscape changes according to the light falling on it, so the same things and events seem to us quite different in our varying moods.

In the instinct of self-preservation, the most fundamental instinct of our nature, lies a tendency to make the individual self the centre of existence. By this principle the pleasure or the pain that is felt, even from early life, must entirely depend on what favors the preservation and the development of our own being. When ideas arise of that which excites pleasure or pain, the instinct of self-preservation stirs, as love or abhorence, and assumes the character of an impulse (Hoffding). When the feeling is determined by the idea of what promotes or hinders self-assertion (self-preservation and self-development) it will appear as a feeling either of power or of powerlessness, according as we think we have or have not at our disposal sufficient means of self-assertion. In his disposition every person has a practical regulator, a level above which his feelings rise only in single instants, and below which it is the exception for them to sink. This constitution of mind, which is due partly to inherited tendencies and partly to circumstances, is the basis of mental integrity. If the feelings habitually transcend these bounds, then there arises in consciousness that vague sense of power, or of fear, or a strangeness of the ego, which is the beginning of a delusional state of the mind. In this connection it may be said that insanity is not a thing apart from human nature. In the main it is but an exaggeration of one or more of the operations of the normal mind, instances of which may be found abundantly in every day experience. Lately while talking of the effects of panic on an audience, a prominent orator in a public address, said: "At this moment were anything to happen so suddenly that we did not quickly understand its purport, we should all go out of these windows." The start one makes on hearing an unexpected noise is a familiar instance of panic, and shows how easily one's sense of fear may be aroused. Only let this state be prolonged and there would be the identical condition which we see in our patients who are suffering from delusions of an exaggerated sense of fear. In the two classes of delusions, viz., those which arise from states of exaltation and those which arise from states of depression you will observe that they are respectively characterized by a feeling of pleasantness and a feeling of unpleasantness. These, as you know, are psychologically the two grand divisions of the feelings, and incidentally the delusions fall into these divisions without any special attempt to make them do so. (See tables.*)

In the study of delusions we examined carefully one hundred and fifty patients; of this number thirty-three men and sixty-nine women had delusions, while seventeen men and thirty-one women gave no evidence of them. Among the men 57.5 per cent. had delusions based on a sense of power; and 78.1 per cent. on a sense of powerlessness. Among the women 44.9 per cent. were based on a sense of power; and 84 per cent. on a sense of powerlessness. These figures agree closely with those obtained at Clarinda and give clear proof of a marked preponderance of delusions based on the sense of inadequacy in the insane. In a number of variable types of insanity, such as dementia præcox, general paresis and manic-depressive insanity, delusions based upon the opposite feelings of power and fear were found to alternate in the same patient; in others, such as paranoia and sometimes general paresis, the two sets of contrasting delusions appeared side by side in the one person, so that the number of delusions was nearly double the number of cases considered. The patients who did not exhibit delusions were composed largely of cases of advanced dementia, in which the mind had become too much enfeebled to form a definite expression of mental content.

According to our observation delusions of altered identity are rare in the insane. Among the one hundred and fifty patients under review no genuine cases of this symptom were found. A patient, whom we shall designate as R. M., was accustomed to refer to herself by the title of King David. At first, her case seemed to fall into this category, but a closer scrutiny of her claim upset the early impression. Having a sense of superiority, she formed the conceit from reading her Bible that she was a modern King David. At the same time she responded to her real name, fully realizing her own personal identity.

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* These are modifications of the tables of Dr. J. W. Wherry, published in the *Alienist and Neurologist*, May, 1904.

In examining patients for the purpose of determining the delusional state of the mind, it is not safe always to regard the expression of every peculiar idea as a delusion. Like children the insane often give vent playfully to phantasy, a tendency to which commonly is largely developed in them. Their delusions are made of different stuff. Strong convictions they are, as we have seen, rooted in the very fiber of their beings. Notwithstanding some apparent exceptions, we found that a delusion always has close reference to self as the center of interest. Contrary to popular belief, an insane person generally reasons well on all topics not related to his delusions, which shows that his judgment here is not at fault. One can neither argue him into absurd notions, nor by ridicule make him an object of derision. He can be made to believe no more readily than his sane brother that the moon is made of green cheese, and can as easily discern a "hawk from a handsaw." It is well known by asylum attachés that every insane patient in a ward sees as clearly as anyone else the evidences of insanity in his fellow companions. In seeking some analogous form of belief which is held as tenaciously and blindly as an insane delusion a good comparison may be seen in the religious faith of a devout Christian, especially if he be simple-minded and illiterate. Ask either one of them the reason of the belief within him and the answer will be in both cases practically the same and equally unsatisfactory. Neither one will be able to tell you more than the bare fact, "I know it, I feel it is true."

According to this theory, then, that an insane delusion takes its rise in disturbances of the vital feelings, we can at once comprehend from what has been said already, that a delusion always begins in a vague way out of the sense of adequacy or inadequacy, or from a morbid feeling of personal identity. The subsequent definite expression of the delusion in the form of a false idea, is simply to be ascribed to an attempt at explanation on the part of the patient.

After all there are, as pointed out by Dr. Wherry and as shown by the tables, but few distinct varieties of delusions possessed by the insane, while the number of false ideas they hold are as numerous as the thoughts of man. When a delusion is shaping itself out of a vague morbid feeling, it is curious to note that the accidental circumstances of the patient's environment generally determine the character of the delusion and control the mode in which it is expressed. To take actual examples, a woman coming down with melancholia, morbidly develops an exaggerated sense of apprehension of harm; a shocking murder takes place at this time in her neighborhood: the vague thought of doing harm appears now in the false belief that she herself has committed the deed. You will observe that the real delusion is not the belief that she had committed the murder, but the vague apprehension that she had done some great wrong. In the case of another woman, who at the time of breaking down mentally passed through an exciting scene at a fire at her home, while suffering no harm from the fire, she soon conceived the idea that she and her dear ones were to be burned alive. Again, a man coming down with acute delirium, having an exaggerated sense of superiority, saw for the first time in a restaurant a device with revolving wings for driving flies from the table. He at once imagined that he had invented the machine and his apparent delusion was that it possessed wonderful powers in many ways and that he was going to make an independent fortune out of it. You have only to turn to your own records to multiply examples, and to confirm the fact that genuine delusions are very limited, while the false conceptions growing out of them are even more numerous than the individual cases.

From the very nature of delusions, they do not frequently change, as usually taught, but remain more or less fixed throughout the course of insanity, be it of long or short duration. The error of mistaking illusions for delusions may easily be made, and one should be cautious respecting the delusion itself, lest its different phases be taken for new ones. The following case may be cited. A woman with vague apprehensions of harm at the onset of her disease, gets from some source the impression that she has poisoned a quantity of meat by which many people have been killed. She is brought to the hospital in an agitated state, and she imagines every man is a detective come to arrest her. Amid new surroundings she soon forgets the poison incident entirely, and then is in dread that she is to be taken out by the nurses as part of a conspiracy and buried alive. After a time this suspicion disappears. Later she confides to a friend the awful secret that she is the evil one himself. A careless clinician in recording these facts in her history might easily draw the conclusion that the patient's delusions are transient and changeable because she appeared to have at least three distinct delusions, viz., poisoning, conspiracy, and evil possession. A closer view of her case reveals the opposite state of affairs. The real delusion, apprehension of injury, remained steadfast throughout, while its mode of expression, the false beliefs, alone was changeable.

Of the one hundred and fifty patients, five men showed disorientation variably as to time, place and environment. The same symptom was displayed in twenty of the women. Illusions of identity appeared twenty-one times—in five men and in sixteen women. Illusions of identity and disorientation of environment is the same symptom occurring in different classes of patients. Illusions of identity are found in delusional patients whose mental tone is good: and disorientation of environment in demented patients whose minds are much enfeebled.

The principal conclusions stated may be briefly summarized as follows:

1. That a delusion takes its origin primarily in a perversion of the vital feelings rather than in a derangement of the intellectual activities of the mind.

2. That delusions may be divided chiefly into those based on the sense of adequacy and the sense of inadequacy with a limited number due to a morbid change of the ego. That delusions of inadequacy largely preponderate.

3. That delusions begin in a vague way out of a disturbance of the vital feelings and that the definite form of the false idea which afterwards appears is the attempt at explanation by the patient to reconcile himself to himself.

4. That the genuine delusions of the insane are very limited in number, but that the false ideas growing out of them are very numerous.

5. That contrary to the text-books, delusions as a rule are not transient and changeable.

TABLE OF DELUSIONS.

TABLE I.

DELUSIONS CHARACTERIZED BY A FEELING OF PLEASANTNESS, BASED ON A SENSE OF ADEQUACY.

I. States of Exaltation.	*A.* Definite (good).	*a.* Body.	Strength. Beauty. Immortality, etc.
		b. Mind.	Ability. Education. Accomplishments, etc.
		c. Possessions	Realty. Personal, etc.
		d. Status.	Personal. Official. Family, etc.
		f. Religion.	Sanctified. Holy sacrifice. Divine mission, etc.
	B. Indefinite (good).	*a.* Superiority. *b.* Possessions. *c.* Emoluments, etc.	
	C. Sexual assertion.	*a.* Divine relations. *b.* Mystic powers. *c.* Superhuman offspring, etc.	

TABLE OF DELUSIONS.

TABLE II.

DELUSIONS CHARACTERIZED BY A FEELING OF UNPLEASANTNESS, BASED ON A SENSE OF INADEQUACY.

II. States of Depression.	*A.* Definite (harm).	*a.* Body.	By poison. By electricity, etc.
		b. Mind.	Thoughts controlled. Faculties destroyed, etc.
		c. Possessions.	Destruction. Robbery, etc.
		d. Status.	Personal. Official. Family. Self-reproach (disgraced, etc.).
		f. Religion.	Persecuted. Self-reproach: Neglected duties. Divine displeasure. Unpardonable sin, etc.
	B. Indefinite (harm).	*a.* Impending calamity. *b.* Vague misfortune, etc.	
	C. Sexual dread.	*a.* Infidelity. *b.* Criminal assault. *c.* "Lost manhood," etc.	

TABLE OF DELUSIONS.

TABLE III

DELUSIONS AND ILLUSIONS OF ALTERED IDENTITY, BASED CHIEFLY ON A CHANGE OF THE EGO.

III. Altered Identity.	*A.* Of self.	*a.* Direct.	*a.* Partial.	*a.* Lost members—head, limbs, etc.
				b. Has another's members—head, limbs, etc.
				c. Has members of unusual material.
			b. Complete.	*a.* Is another person.
				b. Is of unusual material, or animal nature.
		b. Reverse.	*a* Possessed by evil spirits. etc.	
			b. Occupied by animals, etc.	
	B. Of others.	*a.* False recognition.		

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

January 3, 1905.

The President *pro tem.*, Dr. J. Arthur Booth, in the Chair.

A Case of Graves' Disease in a Child.—This case was presented by Dr. J. Arthur Booth. The patient was a girl, 13 years old, whose family history was negative. Her parents were both healthy, and there was no history of any degenerative disease in either branch of the family. The child had suffered from measles, whooping cough and scarlet fever in infancy. She had never had a convulsion. There was no history of fright, nor could any other etiological factor be discovered.

Eighteen months ago, that is, when the child was 11 months old, the exophthalmus was first noticed. Six months later the enlargement of the thyroid became noticeable, and during the past year all her symptoms had become more aggravated. The exophthalmus and thyroid enlargement were very marked, as well as the tachycardia, the pulse ranging between 120 and 140 per minute. In addition to these symptoms there was tremor, general nervousness and insomnia, also occasional headaches.

Dr. Booth said the interesting feature of the case was the early age at which the disease had manifested itself. It was usually observed between the 15th and 25th or 30th year. There were four other children in this patient's family, who were all enjoying good health.

Adenoma of the Pineal Gland, Occluding the Aqueduct of Sylvius, with Escape of Cerebro-Spinal Fluid Through the Nose and Perforation of the Frontal Horn of the Right Lateral Ventricle.—This case was reported by Dr. Adolf Meyer. The patient was a male. In 1894, when diving, he struck the top of his head. Following this, he complained of headache. He became blind in 1898, with chiasma symptoms. In 1899 he had transitory attacks of numbness on the left side of the face, leaving out part of the area of the middle branch. From that time on there was difficulty in moving the lower jaw towards the left. There was no atrophy of the masseter. In August, 1900, cerebro-spinal fluid began to drip from the right nostril, with considerable relief of the general symptoms. On the few occasions when the flow stopped the patient would become sleepy and stuporous for two or three days. During May and October, 1902, the patient, who was then 22 years old, had several general convulsions. It was during that year that he was presented by Dr. Meyer at one of the meetings of the New York Neurological Society. In January, 1904, death occurred in a status epilepticus. The only permanent symptoms had been weakness of the left side of the jaw, a small area of loss of pain sensation of the left lower corner of the mouth, and subjective numbness in the left side of the tongue. The gait remained normal; the knee-jerks were both increased; there was no clonus.

Autopsy: A tumor was found in the form of an adenoma of the pineal gland. It had pressed itself through the roof of the mid-brain behind the posterior commissure, protruding into the third and fourth ventricle and displacing the posterior corpora quadrigemina; the adenoma had practically no sand, and but slight pigmentation. The right lateral ventricle had a funnel-shaped depression in its anterior end, with a perforation through the cortex. In the case reported by Wollenberg (*Arch. f. Psychiatrie und Nervenkrankheiten*, 31, p. 206) there was a tumor of the occipital lobes and perforation of both frontal lobes, but there was no occlusion of the ventricle to help account for the perforation.

In connection with the case reported by Dr. Meyer, photographs made by Dr. C. I. Lambert were presented, and Dr. C. B. Dunlap demonstrated some microscopic sections.

Dr. Joseph Fraenkel spoke of the rarety of these cases, and complimented Dr. Meyer upon his careful and thorough report of the case under discussion.

Dr. Meyer, in reply to a question, said the only motor symptoms the patient presented were those of the masticatory segment, which showed in the deviation of the jaw to the right whenever the mouth was opened. In other words, there was a weakness of the left pterygoid muscles. It was impossible to demonstrate any atrophy of the masseters. There were no motor symptoms referable to the upper extremities.

A Case of Old Fracture-Dislocation of the Spine, with Paralysis, Followed by Recovery; After an Interval of Four Years Gradual Development of a Spastic Paraplegia and Sensory Symptoms, Referable to the Same Level of the Cord.—This case was presented by Dr. J. Ramsay Hunt. The patient was a man 45 years old, a laborer. Twenty years ago he had a venereal sore, followed by a suppurating bubo. He received internal medication for one month. No secondary symptoms were noted. He has had numerous attacks of gonorrhea, followed by the development of strictures. At present a stricture of small calibre exists in the membranous portion of the urethra.

In 1895 he met with a severe accident, falling three stories through an airshaft. He was unconscious for a few minutes, and was taken to Bellevue Hospital in an ambulance. Following the accident he complained of severe pains in the back, and both legs were paralyzed, although not completely, as he could move them slightly in bed. It was necessary to catheterize him a few times after the injury, but this difficulty soon passed away. After the accident an angular deformity of the spine was noted, which still exists. It was located about the level of the twelfth dorsal vertebra, and it was safe to assume that the spine had suffered a fracture-dislocation at that point, and that the spinal cord had been injured at the same time.

After remaining in bed for two months the legs began to improve. This improvement continued, and six or seven months later he was able to resume his occupation as a truck-driver, a laborious one, which necessitated the lifting of heavy weights. One year after the injury he was able to do a full day's work, and was suffering no pain nor inconvenience. He admitted, however, that his legs were not quite as limber nor as strong as they were before the accident. He could not run as fast as formerly, owing to a slight stiffness in the knees. There were no sensory nor vesical symptoms during this period.

For four years or more the patient remained in this condition. He drove a truck, and led the vigorous outdoor life of a laboring man.

About three years ago there developed, very gradually, symptoms of trouble in the lower extremities. He began to complain of sensations of numbness and cold in the feet and legs, accompanied by stiffness and weakness. Upon stooping he felt a numbness in the lower portion of the back, extending down the posterior thighs and legs. During the past two years these symptoms had progressed slowly and steadily without any sudden exacerbation, and without acute pain. The left leg was weaker than the right, and it was important to note that this was true of the initial paralysis and the long period of disability during convalescence.

At the time of the report the patient's condition was as follows: He had a well-marked kyphosis, the tip of which corresponded to the twelfth dorsal vertebra. Corresponding to this there was a posterior bilateral band of hyperesthesia at the same level. The spinal column was fairly mobile, and was not tender to direct pressure or on jarring. There has been no change in the degree of the kyphosis since the accident. There was spastic paraplegia of the lower extremities, with the spasticity and clonus more marked on the left side. Babinski's phenomenon was present on both sides. The skin reflexes were present, and there were distinct objective sensory disturbances in the lower extremities, including touch, pain and temperature, more pronounced on the right side. The man was able to stand fairly well with his eyes closed. He had considerable difficulty in urination, with occasional incontinence. He also complained of sharp, painful sensations and prickling down the posterior aspect of the thighs and legs, and on stooping he felt numb from the hips down. His sexual power was apparently unaffected. Coarse myokymic twitchings were present in the back below the level of the lesion; also in the buttocks, thighs and legs, especially their posterior aspect, after exertion and exposure to the cold. Occasionally during the past year he had sharp, shooting pains in the course of the sciatic nerves, causing reflex movements in the legs; these were more pronounced on the left side.

A Case of Fracture-Dislocation of the Spine Causing a Unilateral or Partial Lesion of the Cord.—This case was presented by Dr. J. Ramsay Hunt. The patient was an elevator operator, 22 years old, who about two months ago fell six stories with his elevator. He was unconscious fifteen minutes after the fall. He was removed to the Gouverneur Hospital, where it was found that he had suffered a fracture-dislocation of the spine, the deformity corresponding to the eleventh and twelfth dorsal and the first and second lumbar vertebræ. In addition there was a fracture of the internal and external malleoli of the left ankle joint.

The case was seen by Dr. Hunt six weeks later through the courtesy of Dr. John Rogers. At that time there was an oval area of anesthesia to touch, pain and temperature over the anterior and lateral surfaces of the right thigh. The right knee-jerk was absent; the right Achilles jerk was present and not exaggerated. The left leg was weak and spastic, with clonus. The Babinski reflex was not elicitable on either side. A girdle sensation was felt in the lower abdominal region, especially on the right side. There was difficulty on urination. Dr. Hunt said the case was a good example of a unilateral or partial lesion of the cord, resulting from a fracture-dislocation of the vertebræ.

The interesting features of the first case were the recovery from the initial paralysis, the long interval of comparative normal function, during which period a laborious occupation was practiced, and then the gradual reappearance of cord symptoms referable to the same level of the cord. The progression of the disease had been most insidious, but always steadily advancing, and of such a nature as to suggest a very gradual compression of the spinal cord.

It seemed reasonable to infer in such a case that the cord was undergoing gradual compression in the spinal canal already narrowed by the fracture-dislocation, and produced by an osseous growth (chronic proliferating osteitis) plus pachymeningeal thickening and adhesions. Spinal syphilis or chronic inflammatory changes within the cord could not be excluded, but Dr. Hunt believed that surgical measures should be taken to relieve the compression.

Dr. Joseph Fraenkel said it was rather difficult to decide the exact nature of the lesion in Case I shown by Dr. Hunt. Pressure on the cord due to an osseous lesion, he thought, would give rise to more severe symptoms than were present in the case, and he suggested the possibility of a post-traumatic hematomyelia.

Dr. Booth agreed with Dr. Fraenkel that an exostosis would probably give rise to more pain.

Dr. Hunt, in closing, said that in studying the case the possibility of a post-traumatic syringomyelia had been considered, as well as syphilis, or a chronic inflammatory condition of the cord, originating at the site of the old injury. The theory that the symptoms followed a hematomyelia, he thought, could be discarded, from the fact that such a lesion was generally found in the gray matter of the cord, and that it would have given rise to dissociated sensory symptoms. In the first case shown it is true there were coarse myokymic twitchings, but no atrophies and no disassociated sensory symptoms. The symptoms were those of a gradual compression of the cord. In cases where the compression was of insidious onset, sharp pains were often absent. In the case shown there were pains of sufficient severity to suggest involvement of the posterior roots.

The differential diagnosis was important in cases. of this character, Dr. Hunt said, because of the treatment. The symptoms were growing progressively worse, and if they were the result of compression, osseous or otherwise, the advisability of an exploratory operation was worthy of consideration. This would be decided, the speaker said, on further observation and after an X-ray picture had been taken.

Diffuse Cauliflower-like Puckering of the Cortex in Arteriosclerotic Epilepsy, or Diffuse Cortical Cirrhosis.—Dr. Adolf Meyer demonstrated a brain that he had obtained through the courtesy of Dr. M. C. Ashley of the Middletown State Hospital. The specimen showed a form of vascular affection, principally of the cortical terminals, in a patient 55 years old. This patient gave a history of having had syphilis at the age of 20. There was mental deterioration for twelve years, and slowly progressive left hemiplegia during the last year of life.

The small vessels of the pia in the affected regions were diffusely occluded. The lesions affected principally the occipital lobes; the temporal and parieto-frontal regions were also affected, leaving, however, intact the convolutions bordering on the Sylvian fossa.

The case belonged to a group of which Dr. Meyer had described two instances in the Pathological Report of the Illinois Eastern Hospital for the Insane, at Kankakee, in 1896. The condition was frequently called diffuse sclerosis, but presented a distinct variety of a progressive disorder of middle life or senescence, akin to the cases reported by Pozzi, Hess, Prout, Blackburn, and probably also by Greiff. The term cirrhosis was used instead of sclerosis because the latter referred more to the broader lesions taking in the white substance, whereas cirrhosis, as applied to the kidney and liver, directed the attention more to the parenchymatous portions, and in this case to the cortex itself.

Dr. T. P. Prout said that in one case he had observed where the early history was obtained, it was found that it belonged to the realm of the infantile cerebral palsies, upon which epilepsy was subsequently ingrafted. This was the light, the speaker said, in which he had during recent years come to regard these cases. The epilepsy was ingrafted upon an early cerebral palsy in the same way as we saw it ingrafted upon other brain lesions.

Dr. Fraenkel said he had seen quite a number of cases in which epileptiform seizures developed late in life, accompanied by some mental deterioration. The diagnosis was of importance, because in a younger person such a condition would be regarded as a general paresis. It was interesting to study the onset of these seizures, which could usually be differentiated from those of ordinary epilepsy by the slight focal symptoms.

Dr. Meyer, in closing, said he did not think it was justifiable to assume an infantile lesion in these cases, because it was possible to demonstrate all degrees of more recent and older foci, with numerous granule cells. In reply to the suggestion made by one of the speakers to call these cases pseudo-paralysis, Dr. Meyer said this would be apt to involve them in the general confusion of that term, which for a while stood for the cases of general paralysis on a demonstrable syphilitic basis, but which we had since learned to recognize as the backbone of that disease.

The following officers were elected for the ensuing year: President, Dr. Joseph Fraenkel; First Vice-President, Dr. J. Arthur Booth; Second Vice-President, Dr. Smith Ely Jelliffe; Recording Secretary, Dr. J. Ramsay Hunt; Treasurer, Dr. G. M. Hammond; Corresponding Secretary, Dr. F. K. Hallock.

PHILADELPHIA NEUROLOGICAL SOCIETY.

January 24, 1905.

The President, Dr. Charles S. Potts, in the Chair.

A Case of Cervical Hypertrophic Pachymeningitis.—This case was exhibited by Dr. William Pickett for Dr. L. C. Peter. Dr. Pickett said the woman had been attending the dispensary of the Medico-Chirurgical College for several years, most of the time under the care of the late Dr. Pearce. Her history is as follows: Four years ago she was confined to bed seven days with an acute illness, which was called peritonitis. Her husband had gonorrhea at that time. In the midst of her illness the woman complained of pain in the arms, back, shoulders and neck. Three months after this attack of peritonitis wasting of the hands was observed. At present there is wasting of the thenar and hypothenar eminences and of the other small muscles of the hand, with consequent flattening of the hand, producing the ***main en griffe;*** also symmetrical wasting of the forearms. The hands are exceedingly weak. Reactions of degeneration are present in these paralyzed parts. Knee-jerks are not altered. No ankle clonus and no Babinski are obtained. There is nothing remarkable in the case at present except the bilateral claw-hand. Evidently there was the painful or meningitic stage of the French writers, followed by the atrophic stage, but there is no supervention of the spastic stage. Dr. Pickett said that recently he had read the paper of Joffroy written in 1873, and it is remarkable how little has since been added to our knowledge of cervical hypertrophic pachymeningitis. It appears from Joffroy's paper that the disease was distinctly described, although not named, by Sir William Gull in 1858.

The apparent gonorrheal origin of this case was the point upon which Dr. Peter had laid stress.

Dr. Pickett said that this case was a legacy from Dr. Pearce's service at the Medico-Chirurgical Hospital, and that he (Dr. Pickett) knew only of the symptoms of the painful stage of the affection from the history and could not say how definite they were. He stated that he had seen one other similar case at Blockley, in which the sensory symptoms were very marked. From the history of the present case one would suppose they were not well marked. It seemed remarkable, too, that there is no involvement of the pyramidal tracts with increased knee-jerks, when the atrophy of the hands has reached such an advanced stage.

A Case of Epilepsy with Myoclonus.—This case was exhibited by Dr. Charles W. Burr. Dr. Burr said he first saw this boy in 1896. At that time he had a history of having had epileptic fits, beginning in the sixteenth month. At first they were very few. He had a series of fits at sixteen months, then an interval of twelve or fourteen months without another, then a few more convulsions and another period without any, and then convulsions occurred quite frequently. When Dr. Burr saw him in 1896 he had three or four attacks of minor epilepsy. He also had a pretty constant twitching of the muscles of the face and shoulders. It appeared then to be the kind of movement seen in habit spasm. Intelligence was good; the boy spoke well and had no palsies, no ataxia, no objective signs of disease except the apparent habit spasm of the head and shoulders, and he had a history of major epilepsy. Dr. Burr did not see him again until last April, when, on going into the ward at Blockley, he saw a boy in bed with violent choreiform movements, in severe chorea. He appeared to be having hallucinations, and told quite a story of having been assaulted by a physician who took him to a building, cut holes in him and sewed them up again. At that time Dr. Burr thought he had to do with a case of chorea with insanity. This diagnosis was incorrect, as 48 hours later he was sitting up perfectly quiet, had forgotten all about the story he had told, and to all appearances was a boy in fairly good health. A day or two after that he had an epileptic fit, which Dr. Burr saw. Since he has been in the hospital he has had eight or ten true epileptic fits. He has also had periods where for days he would have violent spasms of the voluntary muscles, including the muscles of the face; a violent myoclonic spasm lasting for hours or days. Sometimes this movement precedes a fit, sometimes follows one, and sometimes comes on independent of any fit at all. When the movements are bad voluntary movement makes it very much worse. If he attempts to walk he is often thrown down. Again, when the muscular spasm has been very great, for several hours afterward he will not walk. He is too weak to walk. He has no anesthesia, the reflexes are not abnormal, there is no disturbance of speech except the spasm of the mouth and lips, which causes stuttering. If he looks constantly to either side there is persistent oscillation of the eyeballs. At times he is stupid and wants to be let alone, but Dr. Burr had never seen him in any delirium. His skull is somewhat abnormally shaped, especially in the occiput.

Dr. Burr did not believe the case was one of hysteria, as hysteria does not begin at sixteen months of age, nor is it characterized by epileptic fits lasting for many years and showing the symptoms that this boy has. On the other hand, some of his attacks seem hysteroidal in character. He has never had hemianesthesia, reversal of fields of vision, nor anything of that kind. Dr. Burr said he had never seen a picture of disease exactly like this. It seemed to him to be best classified as a case of what Clark and Prout had called palsy with myoclonus. The patient's brothers and sisters were said to be perfectly sound.

Dr. Potts stated that this patient was under his care last summer, and he noticed that the movements grew worse, and indeed often commenced after a period of quiet, when any attention was paid to the boy; also that stern commands would frequently cause them to cease, which led him to believe that there was an element of hysteria in the case. While recognizing that he has epilepsy, Dr. Potts thought some of his symptoms could be attributed to hysteria.

Dr. Gordon had seen this case repeatedly, and stated that the jerky movements would often disappear for a time. The boy had often had falling attacks. He stated that at that time the history of epilepsy was unknown to him, and he thought it was a case of hysteria.

In closing the discussion on his case, Dr. Burr stated that there was concentric contraction of the visual field. He did not like to diagnose hysteria unless it was unavoidable, for it had been his misfortune to diagnose hysteria and afterward find organic disease. He stated that he had done this in cases where he thought there were hysterical convulsions, and which afterward proved to be epileptic convulsions. This boy had a hysterical manner and muscular spasm which varies from day to day, was sometimes controlled by effort of the will, sometimes controlled by voluntary effort, and sometimes made worse thereby. Still, with fits which are known to have been epileptic and a convulsive movement that is not hysterical, and as the case largely agrees with cases reported by Clark and others, Dr. Burr thought this to be a case of myoclonus epilepsy rather than a case of epilepsy plus hysteria.

A Case of Myasthenia Gravis with Paralysis Confined to the Ocular Muscles.—The patient was exhibited and the case was reported by Dr. William G. Spiller and Dr. E. U. Buckman. The case of myasthenia gravis was remarkable in that the weakness was observed only in the ocular muscles. The rapid exhaustion of the levator palpebræ superioris, first of one side and then of the other, especially when both eyes were uncovered at the same time; the recovery after rest, the variation in the paralysis of other external muscles of the oculomotor distribution, the integrity of the inner muscles of the eyeballs, a response of the sternocleidomastoid muscle to the faradic current, suggesting the myasthenic reaction, and the absence of all other signs of implication of the nervous system, make the case probably one of myasthenia gravis of the ocular type.

Notes by Dr. Spiller, Jan. 10, 1905.—The patient had severe headache during ten years, until about six or seven months ago. Vision was not affected when he had the headache, and has always been good.

When he takes off his glasses the upper lids begin almost immediately to droop and continue to do so until the eyeballs are covered, first one then the other. He wears a cover over one eye, and changes this from one glass to the other frequently. When he looks downward both upper lids fall. The external rectus on each side is normal. He cannot look upward or downward very well with the right eye, but can perform these movements with the left eye. He cannot keep both eyes open at the same time more than a minute, but if he covers one eye the other remains open. The iritic response to light and accommodation is prompt in each eye. Sensations for touch and pain are normal in all parts of the body. The muscles of the face, of the tongue and of mastication are normal. The biceps reflex is not unusually prompt on either side. The grasp of each nand is powerful. Voluntary power in the lower limbs is normal. The patellar reflex is normal on each side. Ankle clonus is not obtained. Gait and station are normal with eyes open or closed. The muscles of the facial distribution respond promptly to the faradic current.

Jan 24, 1905.—When he rises in the morning he cannot open his eyes so well as later in the day; formerly this was not the case. He can open his eyes better in the evening. During the entire day be uses his eyes in reading orders in his store (he is a groceryman) or in writing, therefore the disturbance is greater during the day, and is less in the evening, because he lies down.

When he takes off his glasses the upper lid of one eye, depending on which eye he had been using last, begins to fall, and gradually the upper lid falls until there is complete ptosis; while this is occurring the upper lid of the other eye gradually droops until ptosis may be complete or nearly complete on this side. The falling of the upper lid cannot be due to an attempt to overcome the diplopia, because the second lid droops after the first pupil is covered by the lid, and the falling of the lids occurs in the same manner when occasionally he has no diplopia while looking directly forward. He usually has diplopia in looking directly forward, but occasionally he has no diplopia for a few minutes, and then one eye "shoots off," as he expresses it, and diplopia develops. *When the upper lids have fallen, if he closes his eyes for two or three minutes he can open them again,* but with some ptosis of one lid; to-day it is in the left. He cannot keep both eyes open at the same time more than one or two minutes. If he covers the left eye with his hand the right eye stays open better, but after two or three minutes the right upper lid falls. He now can keep the left eye partially open if he covers the right eye. The weakness of the other ocular muscles seems now to be in the elevators and depressors of the right eyeball. The ocular palsy, he says, has seldom been the same at any two examinations. The deltoids react well to a rapidly interrupted faradic current, but there seems to be some exhaustion by this current in the left sternocleidomastoid muscle.

Dr. Sailer stated that he was reminded of a series of cases he saw two years ago. An old woman was brought to his office suffering with an entirely different condition, but she had partial ptosis of one eye and complete ptosis of the other. The ptosis became worse during any form of excitement. The daughter has had the same condition from early childhood. Whenever she became excited it became more pronounced. Her son, representing the third generation, had the same condition. After studying, or when fatigued, ptosis came on, more severe in one eye than the other, but always in both. No definite information could be obtained about her more remote ancestors, but it was supposed that her great-grandparent suffered from this peculiar form of transient ptosis. Two other children of the daughter are normal. Dr. Sailer did not test any case for the myasthenic reaction. None of the patients had ever complained of diplopia.

Dr. Gordon wished to put on record the case of a colored girl who presented a remarkable symptom group, which consisted of drooping of both eyelids. When he talked to her she would open the eyes, but the lids would immediately droop. She could put food in her mouth, but could go no further. Mastication and deglutition were very difficult. This condition persisted for several months, when the patient began to improve. In the case presented by Dr. Spiller and Dr. Buckman the eyelids drooped slowly, while in his case they drooped suddenly. Dr. Gordon regarded his case as one of profound asthenia of a certain group of muscles, probably a symptom of a curable case of myasthenia gravis.

Dr. Eshner thought Dr. Sailer's cases of ptosis were not of the myasthenic type. He mentioned a case of his own which had drooping of the eyelids persisting throughout the man's lifetime, but at that time nothing was known of myasthenia gravis. He also mentioned the case of a colored newsboy. The boy is often seen on the streets of Philadelphia and has ptosis, but probably not of the myasthenic type.

Dr. Mills stated that he had seen several cases of myasthenia gravis, but he had never seen a case of this kind. He mentioned the case of a woman past middle life, seen at the Polyclinic some years ago, who suffered from a peculiar form of drooping of the eyelids. The case was supposed to be hysterical. Hypnotic influence was tried and seemed to do good, but it has seemed to him since that if the case was myasthenic the improvement may have been due to keeping the eyelids closed for some time.

Dr. Spiller, in closing the discussion, stated that there is a condition of congenital ptosis, cases of which have been recorded, and it is possible that congenital ptosis can occur in females. He had seen hysterical ptosis, but thought in most of these cases the condition was one of hysterical spasm. He stated that he had considered the possibility of tumor in the region of the cerebral peduncles, of syphilis, of beginning tabes, of ophthalmoplegic migraine and of myasthenia gravis. He stated that the man was positive that he had not had syphilis. He had been given mercury and iodide in large doses. There is no evidence of tumor; tabes is not probable, as the knee-jerks are normal. There are no symptoms outside of the ocular paresis. Ophthalmoplegic migraine is unlikely because of Dr. Buckman's report on the case. The man is not hysterical and has no evidences of hysteria whatever.

Four cases of beri-beri were reported by Dr. Joseph McCool.

Dr. Pickett stated that he saw the last two of these cases with Dr. McCool, and that without the history of the cases he would have made a diagnosis of multiple neuritis of some other origin. However, dyspnea and tachycardia were still present when he saw the cases. He thought the most striking symptom was one observed in Dr. McCool's third case, namely, ataxia, which was quite marked. The man had to be supported, stepped wide, and put his feet down heel first as in peripheral pseudo-tabes. He had never heard a description of such marked ataxia in beri-beri.

Dr. Weisenburg stated that beri-beri is not a rare disease in the tropics. He had been in Manila when there were about one thousand cases distributed in the prisons. During his two-years' stay there he instituted a number of sanitary regulations which caused a great decrease in the number of cases. In the prisons of the interior there was no beri-beri, while in the prisons along the seacoast it was prevalent. The disease did not differ from neuritis except for the dropsical condition. There are three forms of the disease, the wet form, the dry form and the mixed form. They are all the same except in the amount of fluid present. He stated that there was a considerable degree of ataxia, and that it was rare for a patient suffering with beri-beri to be able to walk. As the patients improve, all show absence of knee-jerk and all become ataxic. In the large number of cases he saw in the Philippines he never saw a case among the soldiers.

The Pathology of Cerebellar Tumors was the title of a paper read by Dr. T. H. Weisenburg.

A Case of Spastic Ataxic Paraplegia Developing After Childbirth.—Dr. S. D. Ludlum reported this case at the December meeting (see p. 334) from the nervous dispensary of the Polyclinic Hospital. The patient, a woman, is married, aged 32 years. Her mother died of tuberculosis, her brother died of Bright's disease, but there was no nervous disease in the family. She had malaria and typhoid fever at the age of 14. She was married at 20 years of age, and has four children living and well, and has had no miscarriage.

Twelve years ago she had her first child. After this for about one month, she says, she had her eyes crossed and had vomiting spells. She then wore glasses, and in three months seemed to be well. The second child was born and she had no disorder following. The third child was born six years ago. When she attempted to get out of bed she found that her left lower limb "gave way" and felt numb, but was not painful to pressure. The general health was good. The weakness and numbness lasted one year and then disappeared. Two years and six months following the birth of this child the fourth child was born, three years ago. She then had a "melancholy spell" of four or five months' duration; at the same time, as soon as she got out of bed she found that there was weakness of both hands (not the arms) and a numb feeling. The left leg was weak. The weakness in the arms disappeared, but continued in the left leg, and about Jan. 1, 1904, both lower limbs became affected. She had no pain, headache nor vertigo, and could hold the urine fairly well and had no rectal disturbance. She was treated at the Woman's Medical Dispensary for pelvic conditions, and was later admitted to the University Hospital, where a blood examination showed a considerable grade of anemia with spastic gait, both legs dragging, toes scraping, and some ataxia, more marked with eyes closed. The patellar reflex much exaggerated on each side. The Achilles tendon reflex was exaggerated on the left side, and lessened on the right side. She had a slight indication of ankle clonus on the right side, but not on the left. Babinski's reflex was distinct on each side, and she had a slight tendency to talipes equino varus. Sensations of touch and pain were slightly diminished in the lower limbs, and she had also subjective sensation of cold in these limbs. The upper limbs were normal, except for slight exaggeration of the reflexes. The cranial nerves were normal.

The eyes were examined by Dr. Shumway. The left nerve had a pathological pallor, the right was of better color, both eyes having refractive error.

She left the hospital somewhat improved, and has since been coming to the Polyclinic Hospital, and has continued to improve on iron and arsenic and tonic treatment, until about Nov. 15, when she says she saw double and was unable to read or thread a needle. Her vision would be good for a short space of time and then return to the abnormal condition.

The eyes were examined by Dr. Posey in December. Paralysis of right inferior rectus and paretic condition of the left inferior rectus and oblique muscles were found. The pupils reacted on both sides, but there was a paralytic condition of accommodation. Both nerves showed pallor on the temporal side, this perhaps indicating a commencing simple atrophy. At present she has very little trouble with her eyes, except that when reading she has a feeling of slight strain.

Strümpell, in a recent article in the *Deutsche Zeitschrift für Nervenheilkunde,* Vol. 27, in writing of spastic spinal paralysis, speaks of the possibility of a beginning systemic spinal disease in women after pregnancy and childbirth. He has seen unusual cases of spastic spinal paralysis which had a relation to childbirth, but as he had no anatomical data, he calls the attention of other observers to these cases.

The possibility of this case presented by Dr. Ludlum being one of multiple sclerosis must be considered, on account of the pallor of the temporal side of the optic disks and the temporary ocular palsies first appearing twelve years ago.

Starr has reported a case of multiple sclerosis developing after childbirth, and Balint has reported a case in which, after four successive confinements, a rapid increase in the symptoms followed.

The long duration of the disease without the development of typical symptoms would make one cautious in diagnosing multiple sclerosis. In the *Lancet* in July, however, are given a number of cases illustrating the frequent occurrence of remissions or recoveries or relapses as a feature of multiple sclerosis.

Müller, in going over nineteen atypical cases, mentions four without either nystagmus, scanning speech or intention tremor.

Periscope

REVIEW OF NEUROLOGY AND PSYCHIATRY

(Vol. 3, 1905, No. 1, January.)

1. A Contribution to the Study of Secondary Descending Degeneration of the Posterior Columns of the Spinal Cord. W. PAGE MAY.
2. Homologies of the Rolandic Region. ALFRED W. CAMPBELL.
3. Some Aspects of Alcoholism. A. HILL BUCHAN.

1. *A Contribution to the Study of Secondary Descending Degeneration of the Posterior Columns of the Spinal Cord.*—The author advances arguments to show his objection to the view formerly held of the structure of the posterior columns of the spinal cord, that were supposed to possess merely nerve fibers which, when they underwent secondary degeneration, always did so in an ascending direction. He describes a set of fibers in the postero-external columns occupying the comma area and the immediate vicinity of the inner margin of the posterior horns; and fibers more mesially in proximity to the postero-median septum. There is also a well-marked tract, wedge-shaped in section lying near the middle line in the columns of Goll throughout the cervical region. This tract consists of short, intermediate and long fibers. As a result of lesions in these areas, secondary descending degenerations occur later in their fibers. But, although the above-mentioned fibers undergo secondary degeneration downwards, there is merely at present a strong presumption that they convey impulses downwards. As for their function, it may be that they serve to couple up movements of the eyes, or movements which have their centers in the mid-brain, with movements of the hands and lower extremities.

2. *Homologies of the Rolandic Region.*—In his paper considering the morphological equivalent of the fissue of Rolando in the lower mammalian brain, the author emphasized the fact that in man and the man-like ape the floor of this fissure forms a sharp dividing line between two territories bearing utterly dissimilar types of cortex, namely, the pre-central and post-central areas. On examinations in serial sections of the nerve cell and nerve fiber architecture of the cortex of the dog and the cat he discovered types closely resembling the pre-central and post-central types of the primate brain. The fibers bearing these types were not divided by the sulcus cruciatus, but by two distinct sulci, one dorsal, shallow and insignificant looking, called the "compensatory state," the other lateral, prominent, and well-known as the "coronal sulcus." The combination of these two is the forerunner of the fissure of Rolando, and so he renounces thecommon belief that the sulcus cruciatus is its antecedent. Giving further historical reasons in support of his objection, he states that the fissure of Rolando acts as a dividing line between motor and sensory cortex. And, if we accept the sulcus cruciatus as the homologue of the fissure of Rolando, the anatomical and physiological demands are not satisfied, as the sulcus cruciatus is deposited in the midst of the motor area, and both of its walls are clothed by one type of cortex. A sulcus in the primate brain interchangeable with the sulcus cruciatus he finds on the oval or paracentral lobule. It is immediately below and in front of the upper extremity of the fissure of Rolando. This fissuret is rarely the same in appearance in any two brains, but it is most commonly found as an isolated, shallow, vertical or oblique furrow.

3. *Some Aspects of Alcoholism.*—The author presents a statistical study of the records of cases of alcoholism in the Royal Infirmary of Edinburgh. The total number of cases of delirium tremens considered was

230, over a period embracing about five years, of this number 209 being men and 21 women. The average age of the patients for both sexes was 39 years. And as to the duration of alcoholism previous to an attack, in men the percentage was 12 years, in women 10 years. The majority of the patients had at least a week's heavy drinking before the attack commenced. The author states that his records afford sufficient evidence to disprove the general view that abstinence after a drinking bout is the cause or an essential factor in the production of an attack. As to temperature, even a moderate degree of pyrexia was not the rule in the cases he studied. And he was also struck with the comparative rarity of acute symptoms of gastro-intestinal disturbance. As regards hallucinations, his records do not bear out the generally conceded idea the smaller the size of the animals seen the worse is the prognosis for the patient. The prognosis of moderately severe delirium tremens is usually good; 16 cases proved fatal out of a total of 230, or 6.97 per cent.

Alfred Glascock (Washington, D. C.).

ARCHIV. FUER PSYCHIATRIE UND NERVENKRANKHEITEN

(Vol. 39, 1905, Part 2.)

16. The Central Gray in Complete Atrophy of the Optic Nerve. Moeli.
17. A Contribution to the Study of Finer Brain Alterations Following Injury to the Skull. M. Dinkler.
18. The Delimitation of Chronic Alcoholic Paranoia. Raecke.
19. Mental Disturbances in Arteriosclerosis and Their Relation to the Mental Diseases of Old Age. Buchholz.
20. History and Critique of the So-Called Psychic Compulsive States. (Conclusion.) Wolfgang Warda.
21. The Theory of Cortical Vision. Erwin Niessl v. Mayendorf.
22. Contribution to the Study of Korsakoff's Psychosis, with Special Attention to the Pathological Anatomy. Gaston Wehrung.
23. Circumscribed Microgyric Formations on the Cerebral Surface and Their Relation to Porencephalus. Miltiades Oeconomakis.
24. Medico-Legal Examination of Sailors. E. Meyer.
25. A Contribution to the Paranoia Question. Siefert.
26. The Relation Between Imbecility and Deaf Mutism. Treitel.
27. Hydranencephalic Twins. Spielmeyer.
28. Contribution to the Knowledge of Epilepsy. J. Finckh.

16. *Atrophy of Optic Nerve.*—In this communication Moeli describes his findings in the gray substance of the third ventricle in a case of total atrophy of the optic nerve. He found a partial disappearance of fibers in the most ventral portion of the wall of the ventricle, and after a careful review of the work done on this anatomical point, he concludes from his own observation that a tract of fibers may be seen coming from the thalamic region and associating itself with the optic tract, which is particularly apparent when the fibers of the optic nerve are atrophied. This tract has nothing to do with that portion of the optic tract related to the hemisphere described by v. Gudden. The minute anatomical details in this paper do not permit review.

17. *Brain Alterations Following Injury.*—Dinkler calls attention to the gradual change in opinion regarding the pathological anatomy of conditions following trauma. He reports two cases with the anatomical findings in which a distinct mental disturbance supervened on a head trauma. A severe mental disturbance following immediately after a relatively slight head injury showed in the first case a high degree of alteration of the vessels of the brain, associated with hemorrhage, softening and cavity formation, with antemortem intravascular bacterial growth, but without

definite signs of gas formation. In the second case, also after simple injury, a high degree of alteration of ganglion cells in the motor cortex was observed.

18. *Alcoholic Paranoia.*—The results of Ræcke's investigation on chronic alcoholic paranoia may be summarized as follows: A chronic paranoia exists which develops on the basis of chronic alcoholism, either primarily or as a direct result of a delirium tremens. Chronic alcoholic paranoia is distinguishable clinically from the classical mental disturbances by certain unimportant traits which are to be referred to the basal alcoholism. The prognosis is very unfavorable, even under conditions of total abstinence. True dementia does not occur. Chronic alcoholic paranoia is to be sharply distinguished from the transitory paranoid excitement which, under some circumstances, may be aroused through repeated alcoholic excesses, quickly disappearing when alcohol is withdrawn. It is furthermore to be distinguished from the terminal mental weakness, in Kræpelin's sense, which remains after delirium tremens or acute hallucinatory mania, and is not capable of further development.

19. Continued paper. Reserved for review later.

20. *Psychic Compulsive States.*—Wolfgang Warda in this paper discusses in great detail, both historically and critically, the various so-called compulsive states, and has attempted on the basis of their etiology and clinical significance to separate the varieties as far as possible. The "Zwangsneurose" he regards as a clinical entity from which are to be separated the specialized varieties of morbid apprehensiveness, the phobias, impulsive insanity, sexual psychopathies and allied conditions. Careful analysis shows such discrepancies between these diseased processes that their grouping under one head is unreasonable and possesses no didactic value. The conception of psychic compulsive states in the sense of a complexus of associated disease processes should be wholly given up.

21. Continued paper. Reserved for later review.

22. *Korsakoff's Psychosis.*—Wehrung, in a deailed article on the Korsakoff symptom-complex, reviews critically our knowledge of this as yet somewhat undefined psychosis, with wide reference to authorities. He reports a typical case of three months' duration, associated with definite symptoms of multiple neuritis. Macroscopically the brain showed only a slight leptomeningitis. Microscopically a parenchymatous neuritis was disclosed, with distinct alterations in the spinal cord and marked disappearance of the supraradial association network of the brain.

23. *Microgyria and Porencephalus.*—Miltiades Oeconomakis believes that microgyria and porencephalia are presumably two stages of a common arterial disturbance. If one hemisphere during fetal life, suffers in development, the sound hemisphere undergoes compensatory changes tending to a diminution of the functional defect. The tænia pontis is to be regarded as an aberrant peduncular bundle, which may also hypertrophy and provide for the motility of the homolateral half of the body in case of an atrophic pyramidal tract.

24. *Medico-Legal Examination of Sailors.*—Since the opening of the psychiatric and neurological clinic at Kiel, which is a main port of entry for war ships, there has been opportunity for the observation of the mental disturbances of sailors, and the duty has devolved upon the clinic of deciding expert questions in relation to this class. The paper consists of a painstaking review of the relatively few cases as yet studied, with the expression of various opinions of medico-legal interest.

25. *Paranoia.*—Siefert, on the basis that a case followed clinically with great care is worthy of publication, presents the report of a man of 46, with minutest details of his mental state. The remainder of the paper is taken up with a critical discussion of the general problem of so-called paranoia.

26. *Imbecility and Deaf Mutism.*—Treitel offers a statistical paper on the relation between imbecility and deaf mutism. The writer discusses the question of consanguineous marriages, and draws such conclusions as the somewhat imperfect statistics permit. It appears that among the Jews, who are notorious for consanguineous marriages, more deaf mutes, idiots and imbeciles occur than among other peoples. Marriages of relatives is considered unwise, and Treitel is of the opinion that such marriages should be at least in a measure forbidden.

27. *Hydranencephalic Twins.*—Spielmeyer, in a primarily anatomical article, adds to our knowledge of early acquired brain defects the description of two interesting brains of twins. The cause of the hydranencephalus which existed was a destructive process due to hemorrhage, which partly led to a breaking down of nervous tissue directly and partly through necrosis resulting from the general circulatory disturbance. Evidence for this was found on the periphery of the defect by the presence of blood pigment. The assumption is made that the hemorrhagic process had nothing to do with an arteritis or a thrombotic or embolic closure of vessels, nor was it the accompaniment of an encephalitis, but rather the result of the extremely thin vessel walls, with anomalies resulting therefrom.

28. *Epilepsy.*—Finckh discusses the general subject of epilepsy on the basis of the histories of 250 cases received at the Tübingen Psychiatrical Clinic, of whom 164 were men and 86 women. This material is worked over from the point of view of etiology, of the relation of infantile convulsions to epilepsy, of the prodromal signs of the epileptic attack, of the epileptoid state, of the course of epilepsy, and of traumatic and late epilepsy. The paper is largely statistical in character, and is of value so far as 250 cases may be useful in drawing general conclusions.

E. W. Taylor (Boston).

PSYCHIATRISCH—NEUROLGISCHE WOCHENSCHRIFT

(Vol. 7, No. 1, April 1, 1905.)

1. Important Decisions in the Field of Forensic Psychiatry. Schultze.
2. The Reichstag's Debate Concerning Mental Defectives in the Army. Konrad Alt.
3. Sleep and Insanity. Georg Lomer.

1. *Legal Decisions.*—These refer to German laws and have no interest for English readers.

2. *Mental Defectives in the Army.*—Calls attention to the fact that mental defectives are often enlisted in the army, and suggests special training of recruiting officers in psychiatry, and a consultation of the school records of recruits.

3. *Sleep and Insanity.*—A short general discussion of the nature of sleep and its relation to insanity. Sleep is never complete; that is, the brain as a whole does not sleep at the same time. Sensations are always flowing in, and where there is sensation there must be mind. This partial sleep has many analogies with insanity; in fact, sleep has been called a "transitory insanity." The cause of sleep is the periodic recurrence of day and night. The waking state is an adaptation to the day; the sleep and dream state an adaptation to the night. To what are the variations of the psychoses an adaptation? This question to be answered must be investigated by the modern "Curve-Psychiatry," which should take up an investigation of the periodicities of circulation, respiration, the effect of thermometric, barometric and seismic variations on the mental state for a better understanding of psychic periodicity.

(April 8.)

1. The Vienna Researches in the Treatment of Paresis. **Konrad Alt.**
2. Important Decisions in Forensic Psychiatry. Schultze.

1. *Treatment of Paresis.*—On the basis of the fact long known that an acute febrile process often influences favorably a psychosis, a number of cases of paresis were treated with Koch's tuberculin, a sufficient dose being given to produce a marked febrile reaction. Sixty-six cases thus treated compared with sixty-six similar cases not so treated gave the following results:

	Cases treated with tuberculin.	Cases not treated with tuberculin.
Living	8	5
Died in first year year	20	39
Died in the second year	23	11
Died in third year	11	6
Died after third year	4	5
	66	66

It will be noted that practically twice as many cases died during the first year among those not receiving tuberculin treatment. Of the number remaining alive those who had received treatment were in much better condition. These results are offered purely tentatively.

2. *Forensic Psychiatry.*—Of no interest to American psychiatrists.

(April 15.)

1. The Legal Basis of State Care of the Insane. Marcus Wyler.
2. Important Decisions in Forensic Psychiatry. Schultze.

1. *State Care.*—A continued article by an English jurist, legal rather than prsychiatric in character. It will not be abstracted.

2. *Forensic Psychiatry.*—Of only local interest.

(April 22.)

1. The Legal Basis of State Care of the Insane. Marcus Wyler.
2. Important Decisions in Forensic Psychiatry. Schultze.

1. *State Care.*—Continued article.

2. *Forensic Psychiatry.*—Not to be abstracted.

(April 29.)

1. Schiller in His Relation to Psychiatry. O. Kern.

1. *Schiller and Psychiatry.*—It is not generally known that Schiller studied medicine and that he was especially interested in problems of the mind. This article is a short analysis of two of his little-known works, viz., "Philosophy of Physiology" and "Relation of the Animal and Spiritual Natures of Man." White.

Book Reviews

THIRTY-FIRST ANNUAL REPORT OF THE MEDICAL DIRECTOR OF THE CINCINNATI SANITARIUM. By F. W. LANGDON, M.D. The Poundford Sta. Co., Cincinnati, O.

This report covers the year ending Nov. 30, 1904, and shows a gratifying degree of progress in this well-known institution. An increase in admissions of 32 per cent., a record of cures of 44 per cent., the installation of a modern card index system in the keeping of clinical records, a complete new clinical laboratory, additional equipments in the medical department and in matters pertaining to the comfort and amusement of the patients, together with plans for a considerable increase in the capacity of the buildings, are some of the features noted in this report.

GOODALE.

DIE HYSTERISCHEN GEISTESSTÖRUNGEN. EINE KLINISCHE STUDIE. Von Dr. Emil Raimann, Assistant der k.k. Psychiatrischen und Nervenklinik des Herrn. Professor v. Wagner in Wien. Franz Deuticke. Leipzig und Wien.

At the present time it has become more or less fashionable to designate hysteria as a psychosis. In this general opinion the authors concur, but it is not a study of the psychotic elements of hysteria that Raimann has considered the subject of hysterical insanity in this monograph, almost the only monographic treatment of the subject extant. He would discuss the more specific insanities having their origin in a hysterical basis. In introducing his study he deals first with the "hysterical character," which has its foundation essentially in a psychopathological heredity, or at times the hysterical personality develops in good stock as a result of head and brain traumata, physical and psychical, in tender years or during adolescence. The type of inducing traumata are worthy of exact observations in this group of cases, and Raimann has laid an excellent groundwork here for further collaboration.

Following many of the French writers, the author finds "suggestibility" as a dominant psychogenic factor. He essays at some length an analysis of the psychological state of suggestibility, but offers little not covered by Sollier, Janet, Raymond and modern French writers.

The clinical differentiation and description of type of hysterical insane states is most fully treated, particularly the analysis of the hysterical dream states. The analogies and contrasts with various catatonic states are illuminating, and serve as useful reminders that many of the conditions diagnosed as catatonic forms of dementia præcox are in reality more allied with the hysterical states so well dealt with in this volume and in Janet's "Neuroses and Fixed Ideas."

In commenting on the chronic types of hysterical insanity the author says that, as a rule, dementia does not take place. In many instances a pseudo-dementia is apparent, and it may be further borne in mind that the hysterical character very frequently develops on the groundwork of a slightly enfeebled mind which dates from childhood.

The etiology of the hysterical "anlage" is dealt with in Chapter IV. The Semitic race seems to shohw a marked predisposition. The Vienna statistics show that women are more often affected than men, the figures showing that proportionately more men are involved in Austria than in Germany, but not so many as in France. Raimann would seek to controvert Freud's teachings concerning the preponderating influence of sexual influences as causative of the condition. In a complete and most convincing summary the well-known effects of the sexual cycles on the mental state are discussed, but the author contends that, while alterations in personality dependent on the sexual functioning, undoubtedly occur, such do not constitute efficient causes for the condition.

Further chapters deal with the Origin of Hysteria, Differential Diagnosis, Prognosis, Therapeutics and Forensic Aspects. A full bibliography is appended.

The work is well worthy a place on the shelves of the student of nervous disorders, be the interests mainly neurological or psychiatric. It is a painstaking and thorough study, differing from most works on the general subject in the psychiatric view-point. JELLIFFE.

News and Notes

Summer Removal of Dr. Smith.—On June 29, Dr. E. E. Smith of South Wilton removes from Kensett, his establishment in Norwalk, Conn., to his summer establishment on Sugar Hall among the mountains of New Hampshire. The removal will be made in a private car attached to the White Mountain Express. This is the tenth year i nwhich this removal has been made, and he has found it of much benefit to his patients. He can receive a limited number of nervous invalids for "a summer in the hills" under experienced medical supervision during the months of July, August and September.

CHANGES IN NEW YORK STATE COMMISSION IN LUNACY SERVICE.

Manhattan State Hospital, East.—James M. Parkinsan, Junior Physician, resignation April 30, 1905, $900 per year.

Manhattan State Hospital, Central Islip.—Calvin B. West, Assistant Physician, promotion May 1, 1905, $83.33 to $100 per month.

Frank Hinkley, Assistant Physician, promotion April 1, 1905, $83.33 to $100 per month.

Alice M. F. Leader, Woman Physician, promotion April 1, 1905, $100 to $108.33 per month.

Kittie Rose Owen, Woman Physician, promotion April 1, 1905, $91.66 to $100 per month.

Fred E. Lettice, Junior Physician, appointment April 3, 1905, $75 per month.

Rochester State Hospital.—Ruth Demarest, Medical Interne, reinstatement May 1, 1905, $50 per month.

Appointment of Dr. Norbury.—Frank Parsons Norbury, A.M., M.D., editor of the *Medical Fortnightly* and Medical Superintendent of Maplewood Sanatorium, Jacksonville, Illinois, has accepted the Chair of Nervous and Mental Diseases, Keokuk Medical College.

Poems of Th. Meynert.—It is announced that Braumüller, of Vienna, will shortly publish a volume of poems of the well-known neurologist Th. Meynert.

Foreign Appointments.—Professor Dr. Aschaffenburg, of Halle, has been appointed as Docent in Psychiatry at the Academy of Practical Medicine at Cologne.

Dr. Paul Schroeder has been appointed Privat Docent in neurology and psychiatry at Breslau.

Dr. Paul Garnier has recently died in Paris.

Dr. J. A. Peters, the Director of the Insane Colony at Gheel, has had the Order of Orange Nassau conferred upon him.

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THE

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OF

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Original Articles

BRAIN TUMORS; A STUDY OF CLINICAL AND POST-MORTEM RECORDS BEARING ON THEIR OPERABILITY AND THEIR SYMPTOMATOLOGY.[1]

By G. L. WALTON, M.D., AND W. E. PAUL, M.D.,
OF BOSTON.

Statistics dividing autopsied cases of brain tumor into operable and inoperable allow the personal equation of the collector so great a range that uniformity of results can hardly be expected. The estimates of the writers who have studied the subject from this point of view vary materially. The average estimate of the various collectors as tabulated by Knapp[2] in 1899 was about 11%. The individual estimates in the table ranged from Seidel's[3] (3%) to Dana's[4] (17%). This difference is doubtless due in some degree to the personal equation.

This element may be lessened by dividing the autopsied cases into three instead of two classes, namely into the operable, the inoperable, and the doubtful, and limiting the designation operable to those cases in which lasting relief might be expected from the operation. Into the operable cases will fall the primary, accessible, well defined tumors which may be removed without cutting into brain tissue. Such tumors spring, in the great majority of cases, from the dura, many of them being designated endotheliomata, a term including the spindle-celled sarcomata and certain psammomata, growths in which, according to Blackburn,[8] groups or whorls of cells have undergone hyaline degeneration and calcification. The modern tendency seems to be to class these tumors according to the nature of the growth so degenerated, for example, spindle-celled sarcomata, rather than according to the form of degeneration. But into the realm of histological classification we should not venture to penetrate.

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[1] Read at the meeting of the American Neurological Association, June 1, 2 and 3, 1905.

[2] Boston Med. and Surg. Jour., Oct. 5. 12, and 19, 1889.

[3] Verhand. d. Deutch Gessellsh, f. Chir., 1892.

[4] British Med. Jour., Oct. 16, 1897.

Into the inoperable class fall the cases generally so acknowledged, such as tumors involving the deeper structures of the brain, the basal ganglia, the pituitary body or the middle lobe of the cerebellum, gliomata extensively involving and replacing brain tissue, as well as most cases of multiple growths and of widespread metastasis.

In the third, or doubtful class, may be placed gliomata and non-encapsulated sarcomata at or near the surface of the brain in accessible regions, many subtentorial tumors, and cysts in which operation can accomplish nothing beyond evacuation of their fluid contents. The prevailing scepticism among pathologists regarding the operability of gliomata seems well founded. Such tumors involve a tendency to faulty growth inherent in the brain substance itself, a tendency to which no boundary can be set, and which can no more be checked than the tendency of fruit to decay after removing the portion obviously decayed, a comparison already rather too favorable for the brain because the difficulty in determining the extent of brain destruction in case of a glioma by exploratory operation is, as a rule, much greater than that of determining the limit of decay in the fruit.

With this classification in view, we have gone through the autopsy records, or studied the specimens, or both, in 221 cases. These cases, with the exception of 15 taken from our own private records, were kindly placed at our disposal for this purpose by the pathologists of the Massachusetts General Hospital, the Boston City Hospital, the Danvers, the Taunton and the Worcester Insane Hospitals, the New York City Hospital, the Montefiore Home, and the Mt. Sinai Hospital of New York City, by the Curator of the Warren Museum, by Dr. Harlow Brooks of New York, and Dr. J. J. Putnam and Dr. E. W.

---

[8] "Intercranial Tumors Among the Insane," 1902. Washington. Government Printing Office.

Taylor from their private collections, and by Dr. Knapp from his recent collection which includes the remainder of the Boston City Hospital cases. The published reports of autopsies found in *Brain* since the establishment of that journal in Blackburn's recent report, and in Knapp's[6] collection of 1891, have been included to bring the total number to 424, a sufficiently large number of consecutive observations to lessen, as far as possible, any selective tendency in the collection. The result of this study is as follows: Out of 424 cases 30, or 7%, were operable; 338, or 80%, were inoperable, and 56, or 13%, were placed in the doubtful class. The combination of operable and possibly operable therefore reached 20%.

Eliminating, on the other hand, the cases in which metastasis or infection was elsewhere present and the cases in which it was stated that no diagnostic symptoms were present, the number of operable cases was reduced to 14, or 3.3%, the doubtful to 34, or 8%. This analysis shows that the highest proportion of operable cases noted in previous statistics (17%) may be approximated in our cases by including the doubtful or possibly operable among the operable, and that, on the other hand, the lowest estimate (3%) may be approximated by including only the undoubtedly operable and excluding those without distinctive symptoms and those with infection elsewhere.

The percentage of possibly operable cases could have been materially increased by accepting the liberal construction of Mills,[7] who cites as probably operable Preston's cerebellar tumor, described as a bilobar tumor, compressing the vermis like a saddle in its inferior part, also exerting some compression upon the quadrigeminum, and apparently having attachments to the callosum, falx and tentorium.

A number of operable growths failed to show localizing symptoms. Many of these were growths near the median line and under the frontal or temporo-sphenoidal lobes rather than in the regions of the convexity usually explored. These facts should lead us to advise exploration over the surface of the brain, particularly in these directions, in preference to following the common method of puncturing the brain substance in various directions in search of a deeply lying tumor. The reason for the continued futility of the latter procedure becomes evident upon the study of a series of autopsies, in which the absence of deep encapsulated tumors is noteworthy. The possibility of the evacuation of a cyst by puncture is of course always present, but this seems the only hope that justifies the procedure, and the hope of permanent relief by such evacuation is small compared with that of finding, for example, an endothelioma by extended search over the surface of the brain. If it should seem advisable and safe to follow both plans in a given case, the surface exploration should certainly precede the tapping.

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[6] "Intercranial Growths." Boston, 1891.

[7] New York Med. Jour., Feb. 11, 1905.

The method of exploration here suggested would probably have changed an unsuccessful to a successful operation in at least one of the cases included in this study, in which an easily removeable endothelioma was found on autopsy, somewhat nearer the median line, in the parietal region, than the site of operation.

It is interesting to note the comparatively simple pathological classification of brain tumors in recent times. Whereas in the early table of Bernhardt[8] 25 distinct varieties of tumor were mentioned, at present nearly all are included under the essential growths of the nervous system (glioma), the connective tissue or non-epthelial growths (fibroma, sarcoma, endothelioma, angioma, angio-sarcoma, osteoma), the epithelial (carcinoma, cholesteatoma), the infectious granulomata (tubercle, gumma and actinomycocis) and the parasitic cysts.

It is interesting to note the infrequency of the diagnosis gumma in recent reports. In fact, this diagnosis does not appear at all in the series of Blackburn, nor does it appear in the Massachusetts General Hospital records since 1896. Multiple tumors of other nature are, however, not uncommon.

These observations are in marked contrast to the frequency with which the clinical diagnosis gumma is made, particularly in case of multiple symptoms. The bearing of this point upon the question under consideration is that the clinical diagnosis gumma should not deter us from advising operation in case of intracranial growth.

Another practical point has been emphasized by this study, namely, the importance of following a fixed plan of operation in spite of previous failures. Two successive autopsy records were found in one hospital in which the symptoms were practically identical and included Jacksonian attacks commencing in the face. Operation in the lower Rolandic region was unsuccessfully undertaken in the first case and not attempted in the second. Operation in the second case in the same region could hardly have failed to disclose and remove, with every prospect of complete recovery, an endothelioma springing from the dura.

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[8] "Beitrage Zur Symptomalogie und Diagnostik der Hirngeschwulste," 1881.

Certain clinical features of cerebral tumor have impressed themselves upon us in this study.

Convulsions.—The clinical records, though often meager, are sufficient to corroborate the fact already established (Knapp, Starr, Mills *et al*) that convulsions in themselves afford little or no help in locating the tumor, as well as the recent dictum of Collier[9] that convulsion, even of the Jacksonian type, is to be discarded as a localizing sign when appearing long after the general signs of intracranial growth. Convulsions accompanied tumors in every situation. The frequency is as follows: frontal 24, cerebellar 15, central convolutions 10, temporo-sphenoidal 11, multiple 7, parietal 6, corpus callosum 5, occipital 4, temporo-parietal 4, pituitary body 3, optic thalamus 2, pineal gland 2, optico-striate 2, centrum ovale 2, temporo-occipital 1, operculum 1, 3d ventricle 1, corpus striatum 2, corpus quadrigeminum 1, lenticular nucleus 1, gyrus fornicatus 1, pons 1.

The frequency of convulsions recorded in cerebellar tumor is of interest in connection with Dana's recent communication. Dana[10] regards sudden fall to one side with loss of sight and consciousness preceded by tinnitus and perhaps accompanied by brief tonic spasm, pathognomic of cerebellar tumor, particularly tumor occupying the cerebello-pontile angle.

Headache and Vomiting.—We have found record of headache together with vomiting in 91 cases, of headache without record of vomiting in 65, of vomiting without record of headache in 11 only.

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[9] "The False Localizing Signs of Intracranial Tumors." Brain, Winter 1904, p. 490.

[10] New York Med Jour., Feb. 11, 1905.

Mental symptoms.—We find mental symptoms in 97 records without including the cases taken from insane hospitals, in some of which, also, the mental symptoms were doubtless due solely to the tumor. The symptoms noted are as follows: loss of memory, stupor, flippancy, delusions of suspicion and of persecution, anxious condition, excitement, dementia, disorientation, abusiveness, viciousness, apprehension, irritability, incoherence, loss of attention, dulness, apathy, forgetfulness, hysterical manifestations, emotional states, hebetude, confusion, amorousness, oddness, fatuousness, lethargy, depression, vacuity, volubility, euphoria, indefference, silliness, delirium, childishness and simple mental failure. It must be remembered that tumor is not necessarily excluded in dementia because of the absence of cardinal tumor symptoms.

Pupils.—Every variety of pupillary disturbance appeared, most commonly sluggishness of reaction. It would seem that little asistance in localization is to be expected from this quarter.

Reflexes.—There is need for an enormous amount of detailed labor before an exact idea can be reached of the location of the mechanisms concerned in the various reflexes and their inter-relations.

The only attempt we shall now make is to add to the existing series of cases showing an appreciable per cent. of diminished reflexes in brain tumor, and to indicate the direction in which this investigation should be pursued,—namely, toward discovering what cerebral lesions cause lessened, and what cause increased, reflexes; and why lesions in some locations cause lessening of reflexes at one period and increase at another. It is noteworthy that the evidence of these cases tends to strengthen the views of those who regard the brain not simply as inhibitive but as playing an active (and in health the principal) part in the production of the so-called reflex phenomena.

In the 123 cases in which record of reflexes was found the knee-jerks were noted simply as *increased* in the following: frontal 8, corpus callosum 5, occipital 4, pituitary body 4, temporo-sphenoidal 3, centrum ovale 3, caudate nucleus 1, cerebellar 3, parietal 2, central convolutions 2, quadrigemini, pons and medulla, multiple, and not stated, each 1, making a total of 39.

The knee-jerks were noted simply as *diminished or absent* in the following: frontal 7, cerebellar 5, pituitary body 4, corpus callosum 3, temporo-sphenoidal 2, occipital, pons, thalamus, centrum ovale, quadrigeminal, and multiple, each 1, making a total of 27. [illegible] case the knee-jerk on the opposite side was at first increased.

The knee-jerk was *increased on the side opposite the tumor* in the following: tumors of central convolutions 5, corpus callosum 4, parietal, cerebellum, pons and medulla, and multiple, each 1, making a total of 13. It was increased in the early history in the thalamus case mentioned above.

The knee-jerk was *increased on the side of the tumor* in one case (thalamus).

The knee-jerk was *decreased on the paralyzed side* in the following: frontal 2, occipital, temporo-sphenoidal and pituitary, each 1, making a total of 5.

The superficial reflexes were noted simply as lessened in 5, cases. The knee-jerks were normal in 50 cases.

The superficial reflexes were noted simply as lessened in 5, *lessened on the paralyzed side* in 7, unequal without further specification in 1, normal in 9.

Lessening of deep reflexes was recorded then in 31 out of 135 cases; increase in 53.

Lessening of superficial reflexes was recorded in 12 out of 21 cases, or more than half.

It is to be hoped that as a matter of routine more complete hospital records will be made in the future than have been made in the past, in order that the value of such records for statistical purposes may be increased.

Even the meagre findings here noted suggest that it is worth while to carry on the study of the brain as an important integral part of the reflex mechanism. The lessening of superficial reflexes in more than half the cases in which these reflexes were noted, certainly tends to bear out the supposition of Grasset that in man the controlling center for these reflexes has risen to the cortex, for if this is true the tendency of all central lesions at whatever height should be to lessen, if affecting them at all. Following Grasset's[11] supposition that the controlling mechanism of the deep reflexes has ascended only to the region of the basal ganglia (particularly the red nucleus, van Gehuchten's tendon-reflex center) there should be, as we have found, great variation in the deep reflexes in cerebral tumors, but including less cases of lost knee-jerk (about 25%) than of increased knee-jerk (about 41%), since the chance of total destruction of the reflex arcs is less than that of interfering with the inhibiting mechanism, which is presumably at a higher level, and perhaps more extensive.

In certain cases of tumor limited to the cortex, the deep reflexes were exaggerated while all the superficial reflexes (unless the Babinski be so classed) were diminished or lost on the paralyzed side and normal on the other. Such cases tend to fortify Grasset's supposition

It is also of interest in connection with this theory to note the proportion of diminished to increased knee-jerks in the central and parietal convolutions, the centrum ovale, and the corpus callosum, as contrasted with that in and below the basal ganglia (corpora quadrigemina, pons and medulla) as well as in the cerebellum.

Classified in this way we find:—

In central convolution tumors the knee-jerk was increased in 7 cases, decreased in none; parietal increased in 3, diminished in none; centrum ovale, increased in 3, diminished in one; corpus callosum, increased in 9, diminished in 3.

Among tumors of the basal ganglia, corpus quadrigeminum, pons and medulla, the knee-jerk was increased in 5, diminished in 3 (in one case of thalamus tumor the knee-jerk was increased only in the early stages).

Among cerebellar tumors the knee-jerk was increased in 4, diminished in 5 cases.

Of tumors in other localities we find frontal 8 increased, 9 decreased; temporo-sphenoidal, 3 increased, 3 decreased; pituitary body, 4 increased, 5 decreased; occipital, 4 increased, 2 decreased.

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[11] Grasset. "Les maladies de l'orientation et de l'equilibre," Paris, 1901, p. 74. See also Revue Neurologique, 1900, p. 737.

Among recent reports Raymond[12] cites a sarcoma pressing on the right peduncle and neighboring corpus quadrigeminum with absence of reflexes of the lower extremities. He regards the phenomenon due not to posterior column degeneration, but to compression of the medulla by stasis of the cerebrospinal fluid. He calls attention to the absence of other symptoms of posterior column disease in case of cerebral tumor, and reminds us that this degeneration was a purely anatomical discovery.

It is of interest in this connection to note that Reh[13] has studied the clinical histories of 100 cases in the literature with reference to the question of lessened and lost knee-jerks in cerebral disease, and the relation of this sign to posterior column degeneration. He explains the diminished reflexes in most cases by the posterior column degeneration (which he does not regard as due to posterior root injury). He explains the fact that the knee-jerk is often normal or increased in spite of this degeneration by variation in seat and degree of the degeneration. Certain cases of varying reflex he attributes to variation of pressure. This is in line with the explanations generally accepted; Batten and Collier,[14] for example, attribute lost knee-jerk in cerebral tumor to posterior column degeneration, but add that if the knee-jerk is normal this does not eliminate posterior column degeneration.

All such explanations start with the assumption that loss of knee-jerk can only be produced by damage to the spinal segments. But is this hypothesis still tenable? The collected observations, clinical, pathological and experimental, seem to point rather to the conclusion that the human mechanisms through which the knee-jerk, for example, is produced, are not simple, and that while the spinal segments in man retain the power to produce the active reflex characteristic of the lower animals, these reflexes in the healthy man are of a different type and represent the composite result of the activity of the spinal paths combined with that of higher paths with which they are in intimate connection. These higher paths are probably in health the controlling part of the mechanism producing the comparatively subdued phenomenon as it appears in the healthy man. To this level may be applied for convenience the term reflex center, provided we recognize the inadequacy of this term in such connection.

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[12] Archive de Neurol., Jan., 1904.

[13] Monat. f. Psych. u. Neurol. Marz, 1904.

[14] "Spinal Cord Changes in Cases of Cerebral Tumor." Brain. 1899, Part 4, p. 473.

It is true that gradual destruction of the higher paths in this mechanism results in exaggerated knee-jerk, a fact which for a long time was deemed adequate to show that the function of the higher path was simply inhibitive; but such destruction does not always produce exaggerated knee-jerk; on the contrary it sometimes results, especially if the destruction is rapid and complete, in a loss of knee-jerk, apparently not always to be explained by the co-existence of spinal lesion.

It is certainly difficult to explain by posterior column degeneration, or spinal trouble, the cases in which the reflex is lessened on the paralyzed side only, that is the side opposite the tumor, to say nothing of the not infrequent temporary loss of knee-jerk on the paralyzed side in the early history of cerebral hemorrhage.

It is true that one case of Finkelnburg, quoted by Reh, suggests that increased pressure of cerebro-spinal fluid may lessen the reflexes. This was the case in which the reflexes, previously absent, returned and became active immediately after lumbar puncture. But even here why should we assume that it was necessarily relief of *spinal*, rather than *cerebral*, pressure that caused the return of the reflexes? But we are not in possession of sufficient data to venture far into the realms of hypothesis. The fact is, however, established that the knee jerk not infrequently disappears in brain tumor, and while confessing our inability to critically analyze all the findings here recorded we submit that they are not easily reconcilable with any theory which accepts the spine as the sole seat of the reflex arcs.

The facts here collected are presented merely as suggestive not as demonstrative or as representing final results. The main object in this branch of the communication is to direct attention to the importance of more complete and careful observations of the locations, and the periods, in which central growths affect the reflexes with a view to furthering the final analysis of the cerebral reflex mechanism.

AN EXPERIMENTAL STUDY ON THE REGENERATION OF PERIPHERAL NERVES.[1]

By S. D. Ludlum, M.D.,
OF PHILADELPHIA,

ASSISTANT PHYSICIAN FRIENDS' ASYLUM, PHILADELPHIA.

The array of evidence is constantly accumulating on the one side, of the multi-cellular origin of the regenerative process, and on the other side of the growth of the newly formed nerve fiber from a central nerve cell. I have made a number of experiments, principally on rabbits, twenty-one of which were successful.

In this paper I have omitted the details of the histological examination in respect to the process of degeneration and regeneration, as these have been well described by many authors and usually in a similar manner. I have given simply an outline of the experimental work and the results obtained.

EXPERIMENTS.

Cases where union had taken place:

12 hours.

Nerve cut entirely through. Axis cylinders do not stain as well in distal portion as in proximal. Some of the methods used have been:—

Weigert Pal stain.

Stroebe stain for axis cylinders.

8 days.

Cut anterior 2-3 of nerve, fibrous union. Stained with Mallory differential connective tissue stain. Some axis cylinders small and contracted, others swollen and vacuolated, all showing signs of degeneration.

5 weeks and 2 days.

Cut entirely through, partial union. There is some debris, degenerated myelin, etc., not yet absorbed and marked infiltration of neurilemma cells. No distinct axis cylinders in the specimens in the distal part.

9 weeks.

Cut out ½ of nerve, leaving a strand to guide union. On killing animal found bulbous union with axis cylinders and myelin sheaths growing through and appearing in distal portion.

10 weeks and 1 day.

---

[1] This work was done at the Sheppard and Enoch Pratt Hospital, Baltimore, Md. Read before the Philadelphia Neurological Society, March 28, 1904.

Cut made entirely through. Very slight union, but axis cylinders were found in extreme distal part. Ink stain brought out fibers distinctly. Arnold's writing ink makes a fairly good selective stain for axis cylinders when tissue is hardened in Müller's fluid. The specimens are cut, placed in ink six days and differentiated as in Weigert Pal.

10 weeks and 6 days.

Cut nerve entirely through. On examination, space between ends was filled with a white scar tissue. This had formed a union and many fibers were seen in the tissue. They decreased in number the further they were removed from the point of union in the distal part.

Black pup: 11 weeks.

Large portion taken out but the space had been bridged over and fibers were easily demonstrated in peripheral portions. Lemon juice and gold make a most excellent stain for embryonic axis cylinders.

15 weeks 6 days.

Nerve cut entirely through. At autopsy the two ends were connected by a thin pale strip of tissue. This proved to be sufficient to convey fibers, as they were easily demonstrated in the distal part.

16 weeks.

Cut entirely through. Good union. Axis cylinders found throughout, myelin not well brought out.

17 weeks.

Slight union. Fibers found in distal part.

18 weeks.

Nerves cut entirely through. At autopsy there was a bulbous union and fibers were found in extreme distal part.

23 weeks.

A good union had taken place and well developed fibers were found in distal segment.

8 months.

Cut entirely through, which resulted in good union with recovery of function. Axis cylinders and myelin sheaths found well developed in distal portion.

Cases where no union had taken place:

6 weeks.

Cut out ⅜ inch. No union and no new fibers in distal portion. Most authors show fibers at fourth week. There are slight remains of degenerated myelin, which is the only change seen from distal portion of experiments many weeks older. The central end always gives the appearance of an endeavor to grow in all directions, many neurilemma cells, and new fibers intermixing and often forming a bulb. Ballance and Stewart claim to show new formed fibers at third week and that they stain readily in Weigert. Weigert and other stains in my cases showed absolutely no fibers where no union had taken place.

6 weeks 3 days.

Cut entirely through; at autopsy, no connection between ends whatsoever, and no fibers in distal segment, though easily demonstrated in proximal segment which ended in a bulbous formation.

7 weeks 3 days.

Cut out ⅜ inch at operation, no union. In distal part there are no fibers whatsoever. In all experiments there are in neurilemma cells lines suggestive of developing axis cylinders, but at no stage have I seen any development of them; they are granular, but never the full length of the cell. There is a strand of nervous tissue, newly formed, running from central end to the muscle continguous to it.

8 weeks.

¾ inch taken out at operation. No union and no vestige of new fibers in distal parts. This was twice the length of time required by Ballance and Stewart to show fibers.

9 weeks.

Cut out ⅜ inch of nerve, no union. No signs of any fibers in distal part, neurilemma cells show no change. A fiber of nervous tissue had grown from central end into near-by muscle.

10 weeks.

Cut entirely through. The union was by a slight band of connective tissue. No nerve fibers in union and none seen anywhere in distal part.

10 weeks.

Cut entirely through, no union. No nerve fibers in distal parts.

13 weeks.

Nerve cut, no union. No fibers found in distal parts.

In these experiments in every case where any union had taken place between the central and distal ends, newly developed fibers could be demonstrated in the peripheral part, but where there was no union between the two parts there was no axis cylinder or myelin sheath in the distal part.

Effort was always made to bridge over the gap and this occurred in thirteen of the experiments.. In the remaining eight where no union took place and there were no fibers in distal part, there was effort to bridge over; and in two cases (one at seven weeks) there was a distinct strand of nervous tissue running from the central end into the surrounding muscle, and in all cases there was formed a bulbous appearance in which were many new fibers intertwining with connective tissue and neurilemma in an effort to burst forth in every direction. No such appearance as this was seen in relation to the distal part. After five weeks there seems to be no change whatsoever in a distal unconnected part, it seems to have been prepared by proliferated neurilemma to form a path for the oncoming nervous process, but showed at no stage any signs of preparing the fiber itself.

Ballance and Stewart, in their extensive work on the subject, claim that the new fibers are formed from these proliferated neurilemma cells, but they are not careful to show whether the two ends may not have joined together.

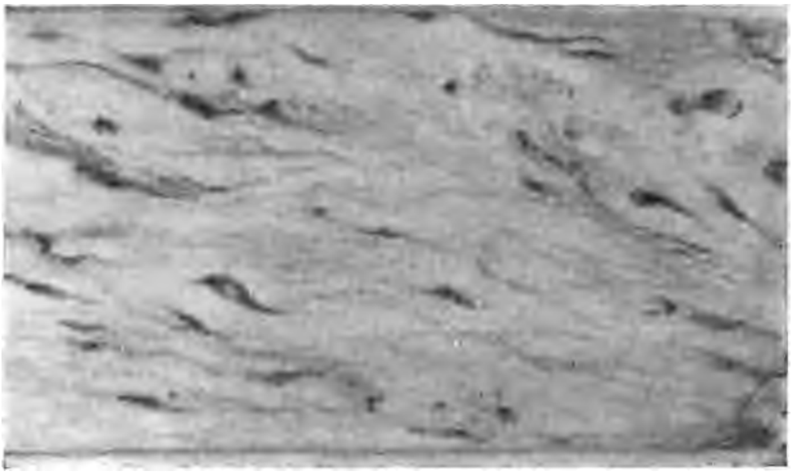
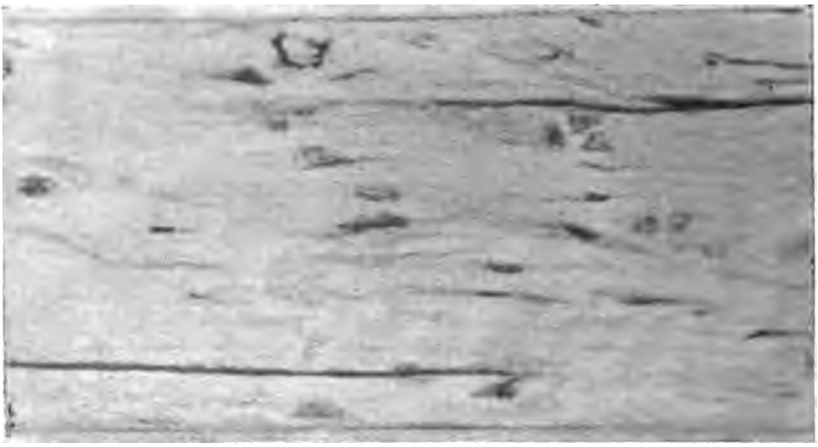


Fig. 1.—Distal portion. No union of two ends of nerve. No new fibers. 13 weeks.



10 weeks.
Fig. 2.—Distal end below union of two ends. Showing growing fibers.

Howell and Huber reached the conclusion that there was no peripheral regeneration. Langley and Anderson,[2] also in this country during the past year working with cats and rabbits, insist that nerve fibers isolated from the spinal cord and spinal ganglia do not reform medulated fibers. In another series of experiments they show that the central end of any efferent fiber can make functional connection with the peripheral end of any other efferent fiber of the same class, whatever may be the normal action produced by the two nerve fibers. They also show that where the peripheral ends of two divided nerves are joined, there is no union. It would seem that if there were peripheral regeneration that these latter would join in some way.

Spiller and Frazier[3] have shown that there is probably no regeneration centralward of posterior spinal roots. This seems to fall in line with the possible idea that neurilemma cells have to be present to prepare a path; but at the same time it does not argue against neurilemma cells being the origin of a multicellular regeneration, for the examples are few of regeneration of the spinal cord where there are no neurilemma cells.

In England, during the last year Mott, Halliburton and Edmunds[4] deny all possibility of the formation of axis cylinders by neurilemma cells. The clinical observations of early return of sensation after suture are not considered trustworthy by them.

At the Oxford meeting of the British Medical Association, last September, Robert Kennedy[5] read a paper in defense of the peripheral regeneration theory. He had examined peripheral segments of nerves up to eighteen months after their division and found the histological characters to have been the same as in young nerve fibers. He implanted a portion of a nerve subcutaneously and in it even found newly formed fibers.

This fact is usually explained as being due to penetration of fibers from the cut end of the nerve seeking the isolated portion. These experiments may be true, but there are many ways of committing error. Kennedy used Stroebe's stain, which is very difficult of certainty. In the Weigert Pal method there is often an arrangement of neurilemma nuclei in a line which would give the appearance of a new fiber, while really it is not, and such arrangements are not uncommon.

Kennedy says if there were such an affinity for nerve fibers to seek out nerve tissue as in a subcutaneous grafting there would be no reason for aiding nature in repairing a divided nerve. We know that it is necessary to suture in order to get good union, and I feel from my own experiments that without union, no matter how distant from a central end of an efferent nerve, there will be no fibers in the peripheral part. Of course there is the careful work of Bethe[6] to be considered, which I do not feel competent to criticize. He worked principally with young animals, however, and did not prove conclusively that it was so in adults. I experimented on a number of a day-old kittens, but they grew so rapidly that I could not be convinced that the experiments were reliable. Another puzzling phenomenon is the return of sensation in two to three days in sutured nerves, which Kennedy claims to be a fact based on forty cases that he has examined. Mott and Halliburton and Edmunds were extremely skeptical regarding the accuracy of such observations.

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[2] Journal of Physics, Vol. 31, 1904.
[3] University of Penna., Med. Bul., June, 1903.
[4] Proceedings of Physiological Society, March 19, 1904.
[5] Brit. Med. Journal, Sept. 24, 1904.

Munzer[7] working in Prague repeated some of Bethe's experiments and found in a divided nerve only the sheaths of Schwann with proliferated neurilemma cells. He demonstrated in an ununited experiment that fibers would follow along the contiguous layer of muscle and enter the distal portion of a nerve. Such a condition as this I found in two cases, where well developed strands of new nervous tissue came from the central end and entered the adjoining muscle but did not reach the nerve.

At the meeting at which Munzer read his paper, M. Raimann said that he had repeated Bethe's experiments and had come to the same conclusion as Munzer.

To my mind it seems improbable that if neurilemma cells originate the new fibrils in the distal and central portions that we have connecting islets of new fibers in the scar tissue where we would not expect to find neurilemma cells.

No one has ever demonstrated a meeting or joining of these islets of so-called new fibers, but they assume that they do. If fibers are seen they would appear to me to be merely parts of fibers from central end cut obliquely in the sections or arrangements of neurilemma nuclei resembling new fibers, for with a Weigert stain they both take the same color.

A most convincing paper is that by Dr. R. G. Harrison[8] in which he demonstrates that in embryos of Rana esculenta the naked fiber is first laid down to the periphey and that the cells of Schwann migrate later from the ganglionic ridge and envelop them. He has observed the movement of Schwann cells from the center to the periphery along the course of a naked nerve fiber in the fin of an embryo. He maintains that the growth of the nerve is by ameboid movement of the protoplasm at the end of the nerve cell prominence as he has demonstrated in various embryos. This work would seem to argue in favor of central regeneration in healing nerves.

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[6] "Allgemeine Anatomie und Physiologie des Nervensystems."

[7] Neurologisches Centralbl., Oct. 16, 1902.

[8] Neiderheim Gesellsch. f. Natur. und Heilkunde zu Bonn, 1904.

PRESIDENTIAL ADDRESS.

THE IMPORTANCE IN CLINICAL DIAGNOSIS OF PARALYSIS OF ASSOCIATED MOVEMENTS OF THE EYEBALLS (BLICKLAEHMUNG), ESPECIALLY OF UPWARD AND DOWNWARD ASSOCIATED MOVEMENTS.

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(*Concluded from page* 448.)

Case 3. Hemorrhage of right tegmentum of the pons.

F. D., aged forty-four years, stone mason, was admitted Feb. 8, 1902, to the service of Dr. Arthur V. Meigs in the Pennsylvania Hospital.

Family history is negative.

Previous history: He had been drinking much for a month before his symptoms developed. Yesterday (Feb. 7) was at work cutting stone and feeling well, when he had a stroke at 4 p.m. He fell to the ground but did not lose consciousness and had strength enough to crawl to a block of stone and sit down. His friends noticed his loss of power on the left side and peculiar inward squint of right eye, which, however, disappeared before he reached the hospital, but returned the following day. He had no real convulsions but shook all over after the stroke.

Present condition: He is a short, chunky Italian and cannot talk plainly, and has loss of power on the right side of the face. Pupils are equal and much contracted. Eyes are straight. Beads of sweat are on the left half of face, which retains the normal wrinkles, while the right side has lost all of these. The tongue can be but slightly protruded, and deviates slightly to the right, and is not tremulous. Respiration is good and even. Fremitus, percussion note, and breath sounds are normal both front and back.

Heart: Pulse is strong and full. Apex beat is not visible. Dulness extends from the left border of sternum to within 2 cm. of nipple line. There are no murmurs.

Liver: Dulness from 5th rib to costal margin in nipple line.

Spleen: 9th to 11th rib and is not palpable.

Abdomen fat and soft.

He has only partial power of the left upper and lower limbs. Ankle clonus is not present. The left patellar reflex is increased, the right is normal. He has no pain and lies in comfort, inconvenienced only by loss of power.

Urine is acid, 1020, trace of albumin, no sugar, many hyaline and granular casts, uric acid crystals.

The patient was seen by me Feb. 17, 1903, at which time I dictated the following notes:

Left pupil is considerably larger than the right and the left eyeball protrudes a little more than the right, and the left palpebral fissure is a little wider than the right. Each pupil contracts promptly to light but soon dilates again. Contraction of the pupils is probably prompt in convergence. Accommodation reaction can not be tested. He cannot close the right eyelids. In looking upward the forehead is not wrinkled on the right side. In showing his teeth only the left corner of the mouth is drawn upward. He has therefore complete or almost complete paralysis in the entire distribution of the right 7th nerve. Cannot protrude the tongue beyond the lips, and it deviates slightly toward the right.

Masseter muscle contracts well on the left side, but much less strongly on the right side, and when the mouth is opened the jaw goes a little to the right. He has therefore weakness of the right masseter and right pterygoids. Apparently he has immobility of the eyeballs, as he is unable to look in any direction other than straight forward. His intelligence is feeble but is sufficient to enable him to understand simple questions.

When his face is stuck with a pin on either side he does not seem to have great pain. If anything, he gives more evidence of pain when he is stuck on the right side.

Voluntary power in the right upper limb is good, while the left is paretic but not completely paralyzed. While he can move the whole upper limb, he cannot raise it above his head or close the fingers firmly.

Tendon reflexes of each upper limb: Biceps and triceps tendon reflexes are not very distinct, and are not greater on the left side than on the right. He gives very little evidence of pain when either upper limb is stuck with a pin. He is weak in the left lower limb, but has still some movement in this limb. He can draw this limb up slightly at the hip and knee, and can move the toes. The movements of the right leg are much better than those of the left, but probably are not fully normal. The patellar reflex is diminished on each side, but is better on the right side. Ankle clonus is not present on either side. The Achilles jerk is diminished on each side. The Babinski reflex is not obtained on the left side, but the toes are neither flexed nor extended. On the right side the movement of the toes is distinctly flexion. Sensation of pain is probably preserved in each lower limb.

On attempting to converge the right eyeball moves down and inward, but the left eyeball moves only slightly downward.

Feb. 20. He has been quiet. Bowel movements have all been involuntary. Temperature has gone up somewhat and breathing is difficult. He has a tendency to let the tongue fall back and obstruct the ingress of air. Hands are twitching at times. Left pupil is larger than the right. Inward squint of right eye continues. Immobility in lateral direction continues.

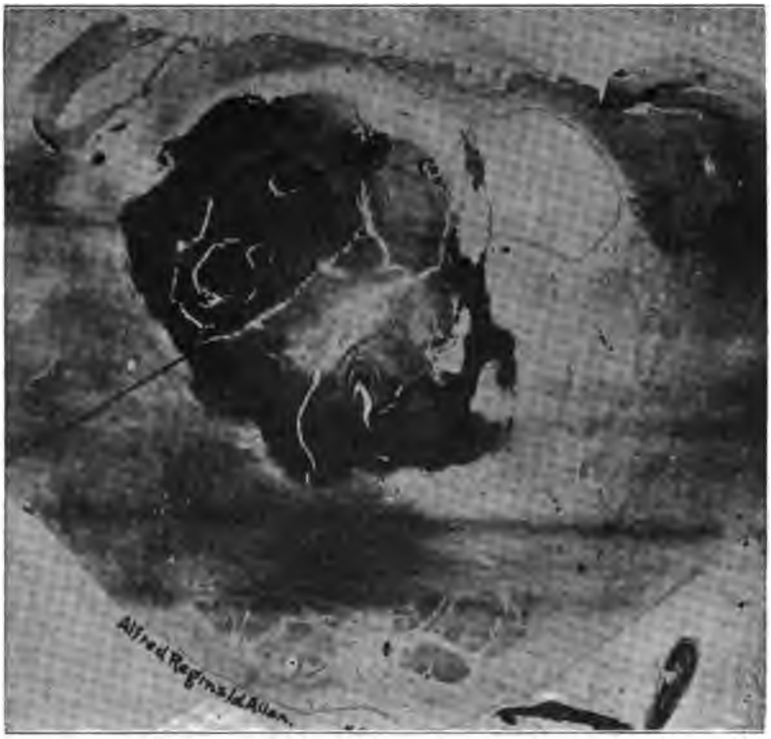

Fig. 9. Line shows hemorrhage of the right tegmentum of pons. Inward deviation of the right eyeball. Immobility of both eyeballs ten days after the apoplexy. Temporary paralysis of all associated movements. Convergence preserved, although weak.

Feb. 22. Condition while very alarming yesterday is now much improved. Breathing is easier, intelligence is better. He recognized and talked with his son.

Feb. 24. The eyes have regained a large amount of lateral movement [the direction not stated]. Hand grasp slightly firmer, pulse moderately good. Temperature touched normal yesterday.

Feb. 26. Condition gradually getting worse, but no special change in nervous symptoms. He is weaker.

Temperature during first few days after admission was between 99 and 101 degrees, then dropped below normal for several days, but on Feb. 20 rose again and remained more or less elevated until death.

Feb. 28. Patient died at 1.30 a.m.

The clinical diagnosis I made in this case was hemorrhage of the right tegmentum of the pons, and it was confirmed by the necropsy.

Important in this case were the following symptoms developing after an apoplexy: Contraction of right pupil, almost complete paralysis in the entire distribution of the right facial nerve, paresis of the right side of the tongue, paresis of the right muscles of mastication, weakness of left upper and lower limbs, but not paralysis, inward deviation of the right eyeball, immobility of both eyeballs ten days after the apoplexy. There was therefore at least temporary paralysis of all associated movements. Convergence was preserved though probably was not normal. It is not probable that the paralysis of upward and downward associated movements would have been persistent if the patient had lived, although the paralysis of right lateral associated movement would probably have remained.

Case 4. This case has been reported by Dr. William Campbell Posey[19] when the disease was in an earlier stage.

The early notes are taken from Dr. Posey's paper.

"F. S., blacksmith, forty-eight years of age, came to the Wills Eye Hospital upon Sept. 22 of this year, on account of failing sight. He had been in attendance at the clinic a year previously on account of supraorbital headaches and general presbyopic symptoms, and had obtained a pair of glasses which gave him normal reading power in both eyes. The examination at that time showed normal fundi; the pupils were noted as being 3½ mm. in size, and the irides responded promptly to light, but sluggishly upon convergence. No note was made of any limitation in the extraocular motions or of any unusual position of the head.

"Corrected vision was normal in both eyes. The patient continued his trade until 3 months ago, when he was compelled to desist on account of attacks of vertigo. These attacks would come on suddenly, without premonitory signs, and were usually induced by change of posture. He had no headache, neither nausea nor vomiting, and his body weight was well maintained. He was mentally depressed, however, most of the time. The patient denies specific infection, but acknowledges masturbation. During 25 years he drank very heavily, being practically in a continual state of intoxication the most of that time; for the past 5 years, however, he avers that he has drunk nothing. He says that he never chewed, but that he has smoked about ½ package of tobacco a week since he was a boy. Although subject to slight rheumatic pain, he has never

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[19] Annals of Ophthalmology, July 1, 1904.

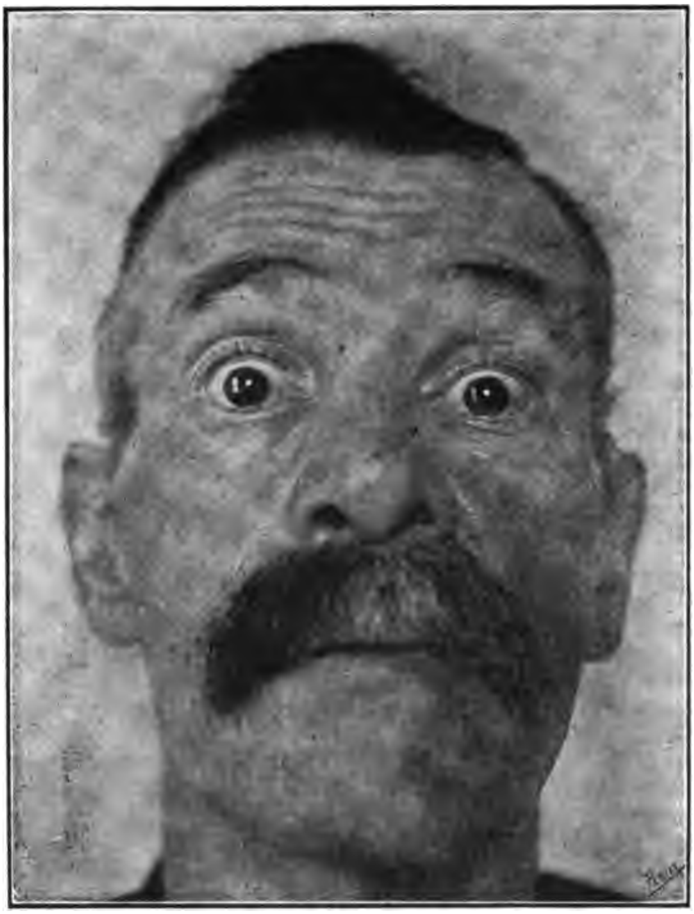

FIG. 10. Paralysis of upward movements of the eyes showing widening of the palpebral fissures and lagging behind of the eyeballs when patient endeavors to look up. (Photograph from Dr. Posey's paper.)

had a severe attack of rheumatism or of any other systemic disease. He has never suffered a traumatism or a fall. His family history is negative.

"While sitting quietly upon the bench, waiting his turn to present himself for examination, the writer noted that the patient presented a peculiar fixity of gaze, which was found to be occasioned bv the fact that the eyes were kept fixed in the primary position and that the patient moved them but little to right or left but sat with his gaze directed straight in front of him. The patient looked anxious and changed the expression of his face but rarely. Closer inspection revealed that the head was thrown slightly back and that several mm. of clear sclera intervened between the margin of the upper lids and the upper limbus of both corneas. When requested to fix the examining finger held in front of the eyes and in the median line, the head was thrown further back and, as the finger was carried upward this was more and more marked, while the eyes remained fixed, not rotating above the horizontal plane. The palpebral fissures grew wider as the patient endeavored to look up (see accompanying photograph) and as the eyes lagged behind, the impression was given of an inverted Graefe lid sign. Lateral motions to the right and left were normal, though the eyes followed the movements of the finger somewhat tardily. Downward motion, too, was conserved, though rotation in this direction was accomplished with an effort and only after several trials. Convergence was lost, the visual axes of both eyes remaining parallel when the finger was made to approach the eyes in the median line; the internal rectus muscle of each eye, however, functioned perfectly in the associated motion toward right and left and also in the unassociated movements inward. In contradistinction to this, however, there were no upward movements in either eye when each was tested singly. The pupils were 3 mm. and were prompt to light but somewhat sluggish to accommodation stimuli. The fundi were normal, the nerves being of good tint. Corrected vision equalled 5—5 in each eye. The visual fields were normal, save for a slight concentric contraction for form and color in each eye.

"When questioned further regarding the nature of the vertigo of which he particularly complained, it was elicited that he had never lost consciousness in the attacks. but that he is unable to stand, on account of extreme giddiness. He frequently has uncontrolable laughing fits at the time. There are no spasms during the attack, and though he falls he is able to rise again at once."

This patient was referred to me for examination by Dr. Posey, Sept. 23, 1903. My notes made at that time are as follows:

There is no contraction of the visual fields tested by the hand. The irides respond promptly to light, but slowly in accommodation. The speech is indistinct and bulbar in character, and he has had difficulty in swallowing during the past three months. The saliva dribbles from his mouth constantly. The tongue is tremulous and possibly a little atrophied on the right side. The fifth and seventh nerves are not involved. Lateral movements of the eyeballs are good. He seems to have some slight impairment of downward movement of the eyeballs, as well as complete paralysis of upward movement. Attempts to look either upward or downward cause dizziness. Sensations for touch and pain are normal in all parts of the body. He complains of bad vision. The breath is very offensive and indicative of mercury. Voluntary power is good in all the limbs. He sways slightly when standing erect with eyes open, and still more so when eyes are closed. The biceps and triceps tendon reflexes and wrist reflexes are probably a little prompter than normal. The patellar reflex is much exaggerated on each side, but the Achilles reflex is normal on each side, and there is no ankle clonus. There is no Babinski reflex, the toes on the left side are not distinctly moved while those on the right side are flexed. There are no urinary disturbances.

Dec. 19, 1903. He has been having severe occipital headache during the past week. The saliva still dribbles from the mouth. When rising from a chair he lost his balance and fell to the floor. He has fallen before this. He does not show hemiasynergy. He says he can hear perfectly.

I received the following note from Dr. W. Zentmayer concerning the patient: "An examination Dec. 28, 1904, showed that the position of rest of both eyes was about 20° below the horizontal plane. All attempted conjugate movements except to the right were lost, and that to the right was limited. When not commanded he at times moves the eyes slightly to the left. The right palpebral fissure is decidedly wider than the left. The pupils are normal. Convergence power is lost."

Through the courtesy of Dr. M. H. Fussell, Dr. Zentmayer, and Dr. P. N. Moylon, I had an opportunity to examine this patient again on Jan. 22, 1905, at which time I made the following notes:

He has no headache and no dizzy spells, but his statements are not reliable. He has not had nausea or vomiting, at least since Oct. 1, 1904. Mentality is affected. Understands what is said to him, but in replying repeats his words several times. His speech is indistinct and he mumbles much in talking, and has not much modulation of his voice. Frequently he utters a peculiar bellowing noise and has forced movements of face, although it is hard to say whether they are more like laughing or crying. He is said to choke frequently in swallowing solids. Raises forehead well on each side and closes eyelids well, and draws up the lower part of each side of his face well. Masseter contracts well on each side, and there is no deviation of lower jaw on opening the mouth. Sensations for touch and pain are normal in the face. The tongue is protuded straight, is not atrophied, and shows no fibrillary tremors, and the movements are free. With finger in right ear he hears the voice when it is not very loud, and same is true when the right ear is tested; he is not deaf.

He is entirely unable to raise the eyeballs, but has slight conjugate lateral movement to the right and left, downward movements are much impaired. Hemianopsia is not present and the vision in upper and lower fields seems to be good.

If left eyelids are closed the right eyeball goes much further to the right, or if the right eyelids are closed the left eyeball goes to the right better than do the eyeballs when he tries to look to the right with both eyes at the same time. Movement of either eyeball separately, when one eye is closed, to the left is also much greater than when he tries to look to the left with both eyeballs at the same time. The associated lateral movements are therefore much more impaired than are the lateral movements of each eyeball separately. Ptosis is not present. Pupils are equal, and irides react promptly to light. Accommodation is not so distinct. Convergence is entirely lost. Movements of head are free. There is some tendency for the eyes to be directed downwards. Grasp of each hand is good. Movements of the upper extremity are good. Biceps and triceps tendon reflexes are exaggerated a little on each side. Sensations for pain and touch are normal in the upper limbs. Finger to nose test shows a little ataxia on each side. Sense of position is normal in each hand. Stereognostic perception is normal in each hand. Resistance to passive movements in the lowers limbs is normal. Voluntary power in the lower limbs is normal at all parts.

Patellar reflex is a little exaggerated on each side. Ankle clonus is not obtained on either side. Babinski's reflex is uncertain because the patient holds the foot very firmly. Achilles jerk is not obtained, also because he holds the foot firmly. He rises from the bed with some difficulty to stand on the floor, and sometimes mades two or three attempts before he can get on his feet. Is able to stand with feet together and eyes closed with very little sway. In walking he does not stagger except occasionally. His gait is very fair, either with eyes open or closed. Frequently in sitting down he looses his balance and falls backwards.

Summary: F. S., forty-eight years of age, (Sept. 22, 1904) had paralysis of upward associated movement with loss of convergence. Downward movement was possible, but was difficult. Lateral associated movements at this time were preserved. The man had been very alcoholic. The speech was bulbar in character, and swallowing had become difficult. The saliva dribbled from the mouth. Ataxia of station was present. Headache was severe at one period. By Dec. 28, 1904, associated lateral and downward movements had become much affected. On Jan. 22, 1905, I examined the patient again and found his mentality poor, speech more indistinct, deglutition difficult, associated upward movements lost, and all other associated ocular movements much impaired. The associated lateral movements were more impaired than the lateral movements of each eyeball separately. The upper limbs were slightly ataxic. Gait was fair, but frequently when he sat down he would lose his balance and fall backwards.

In this case paralysis of upward associated movement developed first, and was followed by paralysis of downward and of lateral associated movements.

Case 5. The report of this case was read by Dr. Zentmayer before the Ophthalmological Section of the College of Physicians.

A. S., male, aged fifty-seven years, butcher, was referred to me by Dr. Zentmayer, Dec. 3, 1904, with the following notes:

"He has had six children, all but two of these died before they were three years of age, one child is still living, aged twenty-nine years, the other died of phthisis. His first wife died of phthisis.

"O. D. 6/ix pt. O. S. 6/xii pt.

"One year ago he had lumbar pain and headache for two days. On the evening of Nov. 29, 1904, after returning from the theater, he found that he was dizzy and that his sight was blurred. He slept well, and the following morning had diplopia (?) on reading, also some nausea and frontal headache. He smokes and drinks to excess. His weight is 200 lbs., and he has not lost nor gained.

"He sits with his head thrown back. The eyes at rest drop slightly below the horizontal plane. There is apparently retraction of the upper lid. The upper lid touches the upper margin of cornea in O. D. In O. S., 1 mm. of sclera shows. On attempting to fix horizontal nystagmus and tremor of inferior fibers of orbicularis develop. Convergence is weak and nystagmic. Lateral conjugate movements are full but accompanied by short nystagmic jerks. Both conjugate and individual upward movement of globes are abolished. At times there is apparently some very slight upward movement of O. D. Right fissure 10 mm. Left 11 mm. Irides react to light and attempted convergence. O. S. iris somewhat sluggish to light. Ophthalmoscopic: peripheral lenticular opacities. Disc red gray with incipient neuritis (?). Veins dark and somewhat engorged. Arteries are a trifle small. Fields, O. D. white contracted to 20°, red contracted to 12°. O. S. white 20° above, 40° laterally, red 10° above, 15° laterally, no scotoma."

Notes made by me Dec. 3, 1904, are as follows:

He has had headache more than a year, and this he describes as a "kind of heavy feeling" in front of head and not a sharp ache. He drinks a large amount of beer and some gin, and smokes much. He has not been getting weak. He has not had any pains in his limbs, except in winter time he has had some rheumatic pains in his hands. He has poor appetite and indigestion.

He has slight drooping of each upper lid, and does not raise his eyes above the horizontal in attempting to look upward, and has then fine lateral nystagmic movements. The movements to the right and left seem to be fully normal, but toward the left he has slight nystagmic movements. Downward movements are normal. The pupils are equal. The reaction of the iris to light and in accommodation is good in each eye, except that the reaction to accommodation in the right eye is not quite so good as in the left eye. He converges with both eyes, but better with the left, and when he converges the upper lids tremble. Hearing for watch is excellent in each ear. The masseter contracts well on each side. There is no involvement of the facial nerve on either side. Sensation for touch and pain in the face and hands and lower limbs is normal. The grasp of the right hand is excellent, the left hand is on a splint because of a recent injury. The biceps and triceps reflexes of each upper limb are not distinct. The patellar reflex is weak on each side, but this probably is because it is impossible to get him to relax his limbs, and it is increased by reinforcement. The gait and station are normal, with eyes open or closed.

On Dec. 29, 1904, Dr. Zentmayer sent me the following note:

"Ten days after you saw A. S. there was noted a marked improvement in supraduction, and 4 days later he complained for the first time of diplopia, and a careful study of the movements of the eyes showed that there remained only a paresis of the left superior rectus. Yesterday all of the muscles (intra- and extra-ocular) seemed to have regained their full power. There is still glycosuria.

Summary: A. S., fifty-seven years of age, (Dec. 3, 1904) suddenly became dizzy and complained of blurred sight. He had used alcohol and tobacco to excess. Convergence was weak and nystagmic. Associated lateral movements were not limited, but were accompanied by short nystagmic jerks. Associated upward movements were lost. Reaction to light was preserved. The disc was red gray with incipient neuritis.

I saw the patient Dec. 3, 1904. He had been having headache. He had slight drooping of the upper lids. He had no signs of implication of his nervous system other than those mentioned.

Ten days later there was marked improvement in supraduction and four days later he complained for the first time of diplopia. There was then only paresis of the left superior rectus. By Dec. 28, 1904, all the ocular muscles had regained their full power.

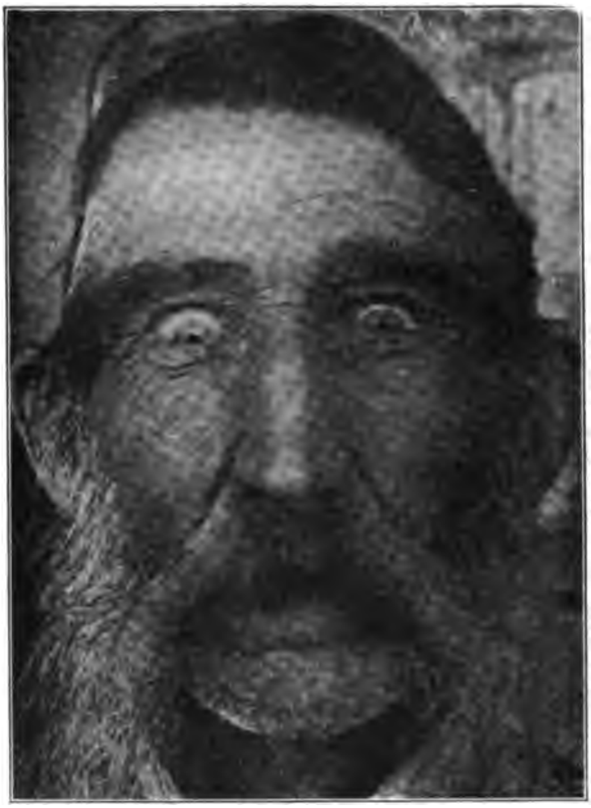

FIG. 11. Attempt to look upward. Forehead is wrinkled, but eyeballs do not move upward. (Case 6.)

Case 6. S. Hea., sixty-three years old, referred for examination by Dr. de Schweinitz, Dec. 14, 1904.

The patient is married and has six children. He attributes his condition to the atmosphere from oil works. He has tremor, especially in the lower limbs. He denies all venereal diseases. He has been very alcoholic and has had difficulty in breathing. Has had headache every night about 8 o'clock, disappearing during sleep. Has bearing down pain across abdomen. Many members of his family have died from cancer.

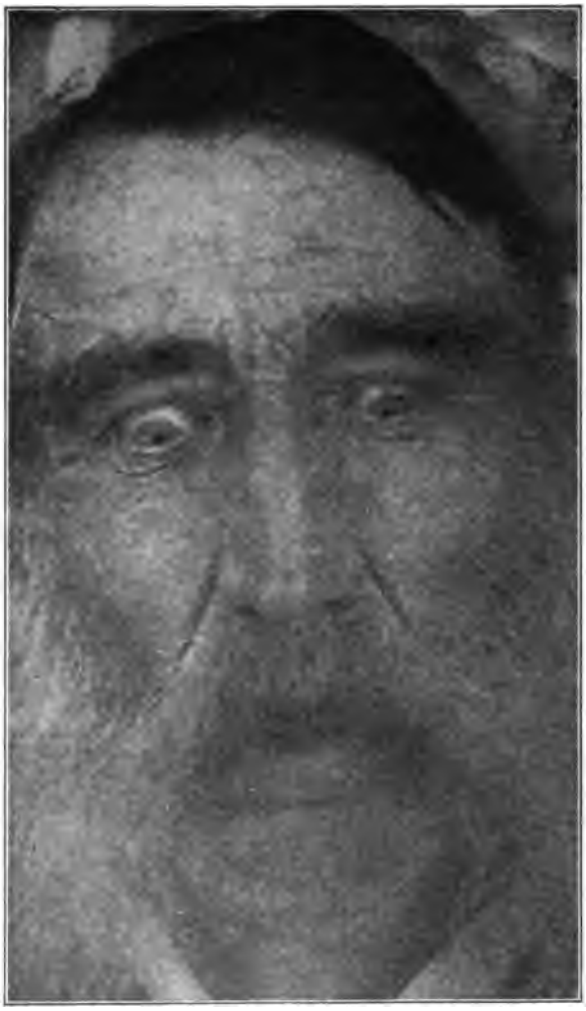

FIG. 12. Downward movement of eyeballs well performed. (Case 6.)

He weighed 181 lbs. a year ago, now weighs 145. No dizziness. Staggers in walking. Is weak in his whole body, "can't carry ten pounds."

He cannot look upward at all, but moves his eyeballs freely in all other directions. There may be some weakness of the right external rectus. The pupils are about equal, reaction to light is present in each eye, distinct but not very prompt. He can converge with each eye. Reaction of each iris in convergence and accommodation prompt and better than to light. No involvement of either facial nerve. Masseters contract firmly. Sensations for touch and pain seem to be normal everywhere. Can hear a watch with right ear only when the watch is pressed against the ear, cannot hear it with the left ear at all. Hears the voice, but says his hearing has become impaired. Soft palate moves freely, tongue normal. Grasp of each hand fair, but not powerful. Biceps and triceps jerks not prompt on either side. Patellar reflex is much diminished on each side. Ankle clonus is persistent on each side, more so on right. Babinski sign is uncertain, but probably present on each side. Sensations for touch and pain normal in the feet. He does not stagger when walking in the room or when standing, with eyes closed or open, but says he staggers on the street. Resistance to passive movements good in all the limbs. The tremor in the right lower limb seen while under my examination appears to be the result of ankle clonus.

Summary: S. H., sixty-three years old (Dec. 14, 1904), had been very alcoholic. He had tremor of the lower limbs, headache, slight ataxia, some subjective weakness of limbs, and complete paralysis of upward associated movements; all other associated movements were normal. Convergence was preserved. The patellar reflexes were diminished. Ankle clonus was present on each side, but Babinski's sign was uncertain.

Case 7. T. Roberts, male, a patient of Dr. de Schweinitz, was examined by Dr. C. K. Mills and myself about Nov. 12, 1904. The man was not ataxic. He could not see light, and had paralysis of upward and downward movements. Dr. de Schweinitz's notes, Nov. 29, 1904, are as follows:

"There is complete blindness, the ocular examinations showing choked disc, beginning to subside with the development of atrophy. He stated that severe headaches had been present before the blindness came on. There was also at times nausea. Headaches have been better, and I believe have been practically absent since the blindness, which he maintains appeared suddenly some months ago. So far as we can determine, there is entire loss of the upward and downward movements of the eyes, therefore failure of associated parallel movement upward and associated parallel movement downward. There is no failure in the power of the eyes to move in associated parallel direction directly to the right, or in a similar direction directly to the left, that is to say, no failure in dextroversion or levoversion. Movement of convergence is present, that is to say, if he is told to look at his own finger, which he rapidly approaches to within a few inches of his eyes, there is convergence of the eyes to this point."

Summary: T. Roberts had paralysis of associated upward and downward movements and was completely blind and had choked disc. He had had severe headache. Lateral associated movements were normal, and convergence was preserved.

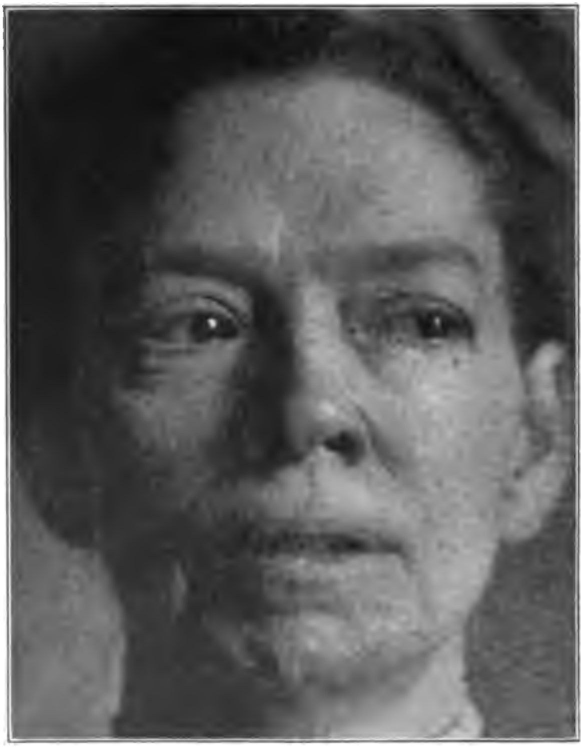

FIG. 13. Looking to the left. Slight weakness of left external rectus. (Case 8.)

Case 8. Mrs. McM., a patient of Dr. Wm. J. McConnell, aged fifty years, was seen by me in consultation with Dr. McConnell Jan. 15, 1905, when I made the following notes. The history and photograph were obtained from Dr. McConnell:

She has had 9 children, no miscarriages. Lungs and heart are normal. Urine has shown albumin and hyaline casts. In Oct., 1904, had her first convulsion, nature of which is not known. Had later another which she says began with numbness in left hand. Since first convulsion has had severe persistent occipital headache, occasionally nausea and vomiting, and she says dizziness. Some ataxia of gait has been observed. Complains that her sight is not so good as formerly. Last Monday (Jan. 9) had a third convulsion beginning, she said, with numbness of left hand, and since then mental state has been a little dull. She has lost flesh.

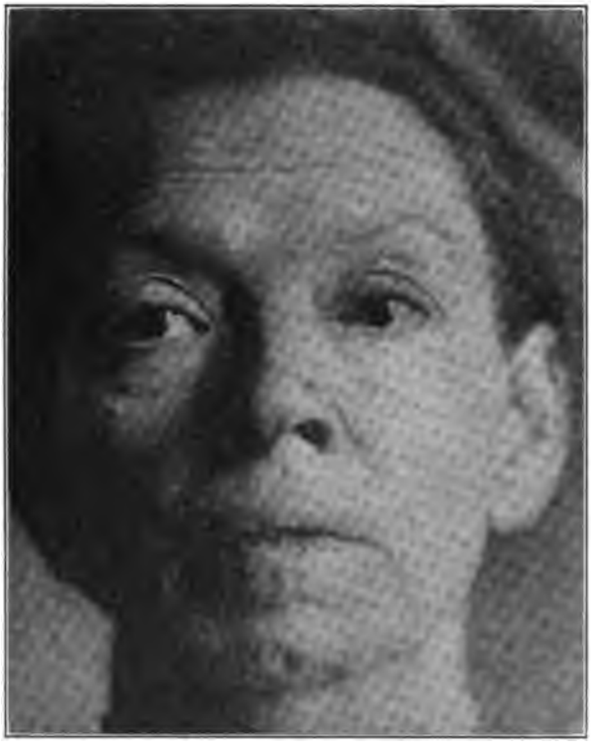

FIG. 14. Attempt to look upward. Wrinkling of forehead in attempt to look upward, but eyeballs do not move upward at all. (Case 8.)

Present condition. Partial ptosis which the patient cannot overcome. Each upper lid droops as low as the center of the pupil when she is looking directly forward. Paresis distinct in upward movement, raises the eyeballs only slightly above the horizontal. Some weakness apparently of left external rectus. Convergence completely lost. No paralysis ot associated lateral movements. Movements to right and downward normal, and usually movements to left normal, but there may be some weakness of left external rectus, and occasionally when left eyeball does not go as far as it should to the left the right eyeball also does not go as far as it should to the left. Slight indication of nystagmus in looking to right. Pupils equal and reaction to light prompt. Movements of 7th and 5th nerve supply normal. Tongue protruded straight and not affected. No deafness to the voice. Sensation for touch and pain normal everywhere.

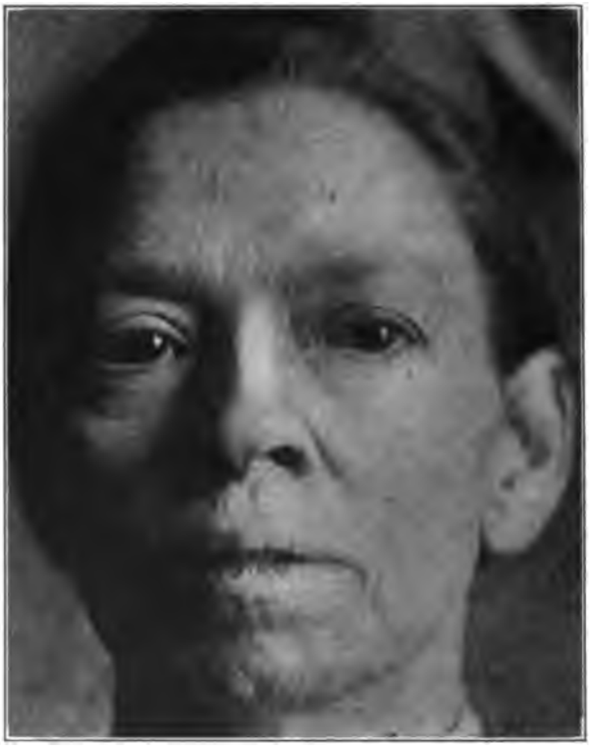

FIG. 15. Attempt to converge, showing power of convergence lost. (Case 8.)

Upper limbs not distinctly ataxic. Biceps and triceps tendon reflexes are not distinct. Movements of the upper limbs are normal. No loss of stereognostic perception nor of sense of position.

Gait slightly ataxic, sometimes falls toward left, sometimes toward right. Patellar reflex not exaggerated. No ankle clonus, Achilles jerks not distinct. No Babinski, toes seemed to be flexed. Voluntary power in all limbs fair, no paresis. No one has ever seen the patient in a convulsion.

I saw this patient again Feb. 1, 1905. The ocular palsies had disappeared, she could raise her eyeballs and upper lids fully, her intelligence was good, she walked well. She had been taking large doses of mercury and iodide. The case appeared to be one of syphilis.

Later the paralysis of associated upward movements returned but disappeared under the same treatment.

Summary: Mrs. McM., fifty years of age (Jan. 15, 1905), had convulsions, headache, nausea, vomiting, dizziness, ataxic gait, failure of sight. I found, Jan. 15, 1905, partial, bilateral ptosis, marked impairment of upward associated movements and loss of convergence; other asociated movements were preserved. The left external rectus was weak. Gait was slightly ataxic.

When I saw this patient again, Feb. 1, 1905, the ocular palsies had disappeared, and she could raise her eyeballs and upper lids fully. She had been taking large doses of mercury and iodide. Later the paralysis of associated upward movement returned and again disappeared under the administration of mercury and iodide.

Noteworthy in this case was the association of partial bilateral ptosis and paresis of the left external rectus with paralysis of upward associated movements; also the disappearance of the ocular palsies twice under the administration of mercury and iodide.

Case 9. W. B. was a patient of Dr. Wharton Sinkler, from whom the following notes were obtained. An opportunity was given to me to examine this patient.

W. B., insurance agent, aged twenty-eight years, was admitted Oct. 10, 1903.

Family history. His mother died from cancer. His father is living. Two brothers and three sisters are living and well.

Previous history. He had measles, pertussis, chicken pox, scarlet fever, diphtheria, and in 1900 syphilis.

He had not used alcohol or tobacco for 5 years. Before that time used both to some extent.

He has worried a great deal about business and felt that he was becoming nervous. May 10, 1903, he developed a right hemiplegia, affecting the arm first, then the leg, although the leg was never completely paralyzed, and he could walk around. Speech was not affected. Oct. 8 he developed diplopia.

On admission he complains of intense headache and diplopia, also of weakness in the right arm and leg, although he can walk and get around fairly well. He also complains of biliousness.

The bowels are regular, appetite has been very poor for the past few weeks, before that time it was very good. Sleep is good.

Heart and lungs are negative.

He has no gastric crises; no shooting pains or girdle sensation.

Reflexes are greatly exaggerated on the right side; slightly so on the left. Dynamometer, R=10. L=65.

The clavicles are very prominent and there is an enlargement of the inner end of the right clavicle. Interspaces are prominent.

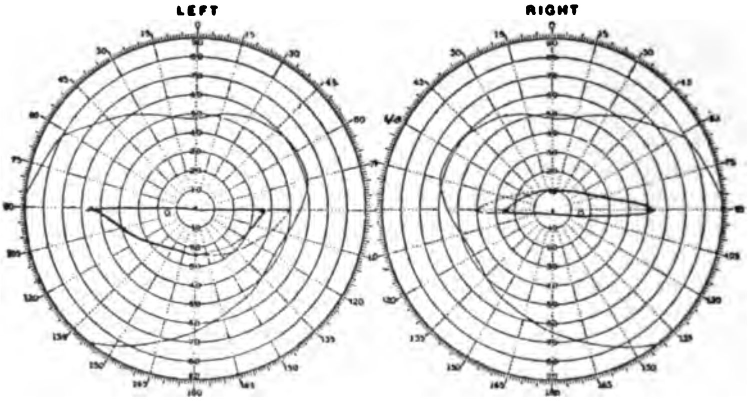

Fig. 16. Upward movement was abolished in the left eye, and was very slight in the right eye. Downward movement was lost in the right eye and was much impaired in the left eye, although it was greater than the upward movement of the right eye. Associated movements were nearly normal in each lateral direction. Convergence movements were lost. Rotation, fields of fixation. Right out = 50, right up = 10, right in = 25-40, right down = 0. Left out = 55, left in = 25-40, left down = 25.

Eyes. Report from Dr. De Schweinitz's clinic book:

O. D. 6/6+40.50, p.p 25; O. S. 6/6+4 O. S. 0.50 p.p 25. Paralysis of accommodation. Lateral outward rotations normal in both eyes; slight loss of inward rotation of each eye. Complete loss of downward movement of right eye, and partial of left eye. Nystagmic movements in both when rotated to extremes of fixation. No choked disc, although slight veiling of right disc; vessels normal. Right pupil—4 mm., faint reaction to light. Left pupil—2 mm., prompt light reaction. No convergence movement, but associated movements are nearly normal in lateral directions.

Nov. 7, 1903. Much improved. Almost all movements restored except downward of right eye, which is still deficient.

Treatment: massage, protiodide gr. 1-6 t.i.d., KI. gr. v increasing.

Oct. 19, 1903. For the past few days the patient has exhibited some mental symptoms, consisting of delusions. He thinks he has recently been married and has a child. That he has a contract to build a depot. That there is a jeweler's shop in the hospital, and that the goods are being auctioned off. At present there is decided mental deterioration.

Nov. 4, 1903. The patient to-day became sick at the stomach and vomited. He also had diarrhea and lost control of the bowels. He is still very much confused, and several times has appeared in the hall without any definite cause. He wanders about. His mental states is unimproved. KI and mercury are discontinued.

Nov. 6, 1903, The patient is discharged to-day somewhat improved. The eyes have recovered considerably from the loss of movements. Speech at present is quite thick and drawling in character. Mental state is poor.

Summary: W. B., aged twenty-eight years, had contracted syphilis in 1900. Right hemiplegia developed May 20, 1903, and in October of the same year he had diplopia. Headache became intense. Upward movement was abolished in the left eye, and was very slight in the right eye. Downward movement was lost in the right eye and was much impaired in the left eye, although it was greater than the upward movement of the right eye. Associated movements were nearly normal in each lateral direction. Convergence movements were lost. The pupils were unequal, and reaction to light in the right eye was faint, but prompt in the left eye. Optic neuritis was not present, but there was a little veiling of the right disc. Each ciliary muscle was completely paralyzed. Convergence was lost.

By Nov. 7, 1903, the patient was much improved, and almost all the ocular movements were restored, except the downward movement of the right eye.

ABSTRACTS OF THE CASES OF PARALYSIS OF UPWARD OR DOWNWARD ASSOCIATED OCULAR MOVEMENTS REPORTED BY THE LITERATURE.

The original papers, with three exceptions, were obtained and the abstracts were made by me from these.

Henoch[20]: The eyes were turned downward, and the child could not raise the eyes upward. Lateral movements were normal. Pupils were somewhat dilated and reaction was slow. Later were vomiting, drowsiness, paresis of the right arm, irregular pulse; still later, complete paralysis of the right limbs and right side of the face, deviation of the right eye inward. The lesions were tuberculous meningitis and tubercle of left posterior part of the corpora quadrigemina. It is not stated whether the third nerve nuclei were normal.

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[20] Henoch. Berliner klin. Wochenschrift, March 21, 1864, p. 125.

Wernicke[21]: After an apoplexy the symptoms were left hemiplegia, great limitation of upward and downward movements of the eyes, with normal lateral movements. There were no ptosis and no changes in the fundus. An apoplectic focus was found in the right optic thalamus, lenticular nucleus and anterior part of the right side of the corpora quadrigemina. The oculomotor nucleus was involved on the right side.

Priestley Smith[22]: Case 4. The symptoms were headache, vomiting, vertigo, diplopia. The eyes maintained a constant convergence toward a point two feet distant from the face, and the patient was unable to diminish to the smallest extent the convergence, nor could he increase it very much. Vertical movement upward above the horizontal plane was totally lost. Both eyes followed an object to the normal extent to the right or left. The patient steadily improved and he became able to look upward, but the ocular symptoms returned and the parallel conjugate movements were less free than at the former examinations. Tested by the horizontal and vertical movements of an object before the face, each eye was found to have the following range:

Outward, only a little beyond the median line, and much unsteadiness and jerking attended the effort.

Inward. to the full extent, but with a little jerking.

Downward, apparentlv to the normal extent.

Upward, no movement was possible above the horizontal line, and much jerking attended the effort. There was no necropsy.

Case 5. The symptoms were headache and vertigo, followed by sudden unconsciousness lasting one day, and supposed to be hysterical. One or the other eye deviated inward. The outward, inward and downward movements of the eyes were nearly or entirely to the full extent, but upward movements were not more than half the normal extent. The ocular symptoms later disappeared.

Nieden[23]: The symptoms developed suddenly. The patient, a man aged 45 years, became blind while bending over. Examination showed that vision was much impaired. The eyeballs could be moved promptly downward, outward or inward, but not upward beyond the horizontal line. When the attempt was made to look upward the eyeballs were drawn backward and forward in a peculiar manner. There were no fundus changes. The lesion was supposed to be a cerebral hemorrhage. Recovery in this case was complete.

Gowers[24]: The symptoms were headache, vomiting, weakness of the legs, a few convulsions. The patient's manner was suggestive of hysteria. She had well-marked double optic neuritis. The eyes moved freely in all directions but upward. When she tried to look upward the eyes moved very little or not at all above the horizontal line. The pupils were equal, 4 mm. in diameter, acting very little to light and not at all on attempts at accommodation. The patellar reflexes were much exaggerated. A very small tumor was found in the middle line behind the posterior quadrigeminal bodies, damaging these slightly, the velum and the adjacent part of the inferior vermiform process of the cerebellum.

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[21] Wernicke. Berliner klin. Wochenschrift, July 3, 1876, p. 394. "Lehrbuch der Gehirnkrankheiten," Vol. 2, p. 84.

[22] Priestley Smith. Ophthalmic Hospital Reports, 1876, Vol. 9, p. 22.

[23] Nieden. Centralblatt für prak. Augenheilkunde, July, 1880, p. 209.

[24] Gowers. Transactions of the Ophthalmological Society of the United Kingdom, Vol. 1, 1880-81, p. 117. A Manual of the Diseases of the Nervous System, Sec. Edition, p. 185.

Parinaud[25]: Case 4. A man, 67 years of age, had had attacks of polyuria and heaviness of the head 18 months. In one of these he had difficulty in standing and the vision was disturbed; he had a tendency to fall to the left. At the same time he had complete paralysis of upward and downward movement of the two eyes, except that the right eye could be moved upward a trifle. Movements to the left and right were normal. Convergence was lost. The upper lids were normal. The pupils were moderately contracted and a little unequal. The reaction to light was lost. The visual fields and central vision were normal. The pulse was slow. No necropsy was obtained.

Case 5. A woman, 20 years of age, had had headache, especially on the left side, about a month. Diplopia suddenly developed. A month later she was found to have complete paralysis of convergence. Lateral associated movements were normal, as were also downward movements, while upward associated movements were almost lost. Crossed diplopia was present. There was no lesion of the fundus, and no amblyopia. Several times numbness of the left lower limb occurred, and was followed by some weakness.

Ormerod[26]: A man, aged 44 years, had paresis of the upward movement of the eyes, most marked in the right eye, especially when the patient looked to the right. There was vertical nystagmus when he tried to look upward. The other ocular movements were normal. There was no definite neuritis. The symptoms had been present ten months, but he had had a similar condition fifteen years previously.

Otto Hope[27]: The symptoms were: Headache, general tremor occurring in attacks, right-sided deafness and vomiting. The patient was unable to raise his left eye above the horizontal, and the right eye he could raise only slightly; the downward movement of the eyes was much impaired. Lateral movements were normal. He had reflex rigidity of the pupil, and slightly choked discs. The limbs were not paralyzed. A tumor the size of a pigeon's egg was found in the corpora quadrigemina; the aqueduct was preserved only in the posterior half. The tumor was a telangiectatic sarcoma.

Reinhold[28]: The symptoms were vertigo, headache, paresis of each external rectus, vomiting, disturbance of vision without optic neuritis, bilateral ptosis and limitation of upward movement of the eyes, with nystagmus. A gliosarcoma of the pineal gland was found. The corpora quadrigemina and the aqueduct of Sylvius were compressed, but the tumor was easily separated from the former.

R. Thomsen[29]: The symptoms were failure of memory and intelligence, headache, vertigo, weakness, tremor, rigidity of the limbs, small and unequal pupils, iritic reflex to light lost on the right side and weak on the left, pallor of discs. Both eyes could be moved to the right, left or downward, but with nystagmus. The eyes could scarcely be raised above the horizontal, and the right was more affected. A gumma was found at the exit of the oculomotor nerves and growing into the peduncles and implicating the nucleus ruber, and especially the fibers of the right oculomotor nerve within the peduncle. Ependymitis diminished the size of the aqueduct of Sylvius considerably, but the gray matter about the aqueduct and the oculomotor nuclei appeared to be normal. Nissl's method was not employed. There seems to have been no compression of tissue by the gumma.

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[25] Parinaud. Archives de Neurologie, Vol. 5, No. 14, 1883, p. 145.

[26] Ormerod. Brit. Med. Journ., March 22, 1884, p. 564.

[27] Otto Hope. "Ueber einen Fall von Tumor der Vierhügel," Inaug. Dissertation, Halle a S., 1888.

[28] Reinhold. Deutsches Archiv. für klin. Med., Vol. 39, 1886, p. 1.

[29] Thomsen. Archiv. für Psychiatrie, Vol. 18, 1887, p. 616.

Nothnagel[30]: A male, 24 years old, presented the following symptoms: Headache, diplopia, paresthesia of left limbs, epileptic attacks, choked discs, paresis of the left upper and lower limbs, ataxia of the left upper limb, and objective disturbance of sensation in the left upper and lower limbs and left side of face. The left abducens was paretic. Upward and downward movements of each eye were much impaired. Lateral movements, except outward movement of the left eye, seem to have been good The left facial nerve was paretic for voluntary movements. There was no necropsy at the time of the report.

Samuel Gee[31]: The patient was six years of age. The symptoms were loss of appetite, vomiting, headache, ataxia, convulsions followed by inward squint of the eyes lasting three days, then the eyes began gradually to turn upward. About the same time the power of the limbs was lost. The head was retracted. Both eyes were turned upward, so that the corneæ were concealed by the upper lids. The right eye also turned a little outward. There was constant vertical nystagmus, no ptosis. The discs were white, their margins blurred and their vessels small; *i.e.*, they were atrophic and probably secondarily to optic neuritis. Palsy of the limbs was very incomplete, and there was no rigidity.

A pulpy, gelatinous mass was found over the surface of the cerebellum above and behind the medulla oblongata, a large, bleb-like sac distended with fluid bulged out between the crura cerebri. The corpora quadrigemina were flattened and "distended over a mass of gelatinous pink new growth which formed the anterior projecting extremity of a mass of similar new growth filling the whole cerebellum." The growth was considered to be sarcomatous.

Evidently the eyes could not be directed downward in this case, because it is stated that it was difficult to get a view of the fundus on account of the position of the eyes.

Eisenlohr[32]: The symptoms were drowsiness, vertigo, later tremor of the left arm, dilatation of the right pupil, slow reaction of the pupils, no changes in the fundus; still later, limitation of movements of eyes, especially upward and downward, polyuria, headache, vomiting, stupor, ataxic gait; still later, choked discs, right-sided ptosis. A bullet wound was found in the right side of the corpora quadrigemina. A portion of the third nucleus was injured.

Lichtheim[33]: Case 1. In addition to the signs of brain tumor, one abducens became paralyzed, and was followed soon by paralysis of upward movement of both eyeballs, optic atrophy and rigidity of the pupils. A glioma occupied the whole of the corpora quadrigemina and had grown into the right optic thalamus.

Case 2. The symptoms were vertigo, ringing in the ears, paralysis of one abducens and paralysis of upward movement of both eyeballs. Choked discs were present, and later ataxia. The tumor was in the corpora quadrigemina and optic thalamus.

Case 3. The symptoms were headache, diarrhea, vomiting, ataxia and weakness of the right side of the body. The left abducens was paralyzed, and there was paralysis of upward movement of the eyeballs and optic neuritis. A caseous tumor was found in the corpora quadrigemina, not involving the anterior part of this structure.

The abducens paralysis was attributed to pressure upon the nerve at the base of the brain, and was present in all three cases.

Parinaud[34]: The patient, a man, became paralyzed in the left side of the face and in the left upper limb. The lower limb seems to have escaped, and the weakness of the left upper limb was of short duration, except in the fingers. He was almost completely unable to look upward, and lateral movement to either side was very imperfect. He was able to look downward without difficulty. Convergence was normal. The pupils were of normal size, and the iritic reaction was normal to light and convergence. There was no lesion of the fundus. He had partial left homonymous hemianopsia. Parinaud localized the lesion in the cortical center of the face and adjoining region.

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[30] Nothnagel. Wiener med. Blätter, 1889, No. 9, p. 131.

[31] Gee. Saint Bartholomew's Hospital Reports, Vol. 26, 1890, p. 106.

[32] Eisenlohr. Münch. med. Wochenschrift, May 20, 1890, p. 364.

[33] Lichtheim. Deutsche med. Wochenschrift, Nov. 17, 1892, p. 1043

[34] Parinaud. Annales d'Oculistique, 1892, Vol. 107, p. 283.

It is noteworthy that in this case reported by Parinaud himself convergence was normal.

Verrey[35]: The paralysis of associated upward and downward movement of the eyes developed suddenly, and was associated with a tendency to deviation to the left in walking. The symptoms lasted only a few weeks. A small hemorrhage was supposed to have occurred in the corpora quadrigemina or near this region.

Sharkey[36]: The patient, a man aged 43 years, became stuporous, deaf in the right ear, numb in the right half of the body, had right hemianopsia and paralysis of the superior rectus in each eye, and of the inferior oblique of the right eye. Both pupils reacted to light and accommodation, but sluggishly. There was no optic neuritis. Sensation was affected in the right side of the body. Speech was thick. Mr. Nettleship found that there was no movement of the eyes upward beyond the horizontal, and that downward movement was also defective, especially in the right eye. Concomitant lateral movements were full, but convergence was very defective, especially in the right eye. Irregular nystagmus was present in lateral and in upward movements. Fundi were normal.

Later, loss of power was noticed in the right side, and optic neuritis developed. A tumor was found in the corpora quadrigemina and upper part of the left cerebral peduncle.

Sauvineau[37]: A woman, aged 73 years, had an apoplectic attack with transitory right hemiplegia at the age of 70. Three years later she had another apoplectic attack with slight right hemi-paresis, and at the time of examination by Sauvineau she was a little weak in the right side. She did not have strabismus nor ptosis, and lateral movements of the eyeballs were normal, although they were associated with a little rotary nystagmus. Upward movement was lost in both eyes, and downward movement was feeble and associated with vertical nystagmus. Convergence was lost. At times she had diplopia. The pupils were equal and of normal size, and reaction in accommodation was normal. The light reflex was preserved, but feeble on the right side, and abolished on the left. No fundus changes were present, and the visual fields were normal. There was no hemianopsia. Sugar was present in the urine.

Schroeder[38]: The case was remarkable, as the paralysis of associated movement was downward. As cited by Teillais, a man 31 years old, was suddenly attacked with fever, headache, vomiting, convulsions of the limbs, complete ophthalmoplegia and amblyopia. After four or five weeks most of the symptoms disappeared, but there was complete paralysis of associated movement downward, and the eyes could not be moved downward below the horizontal. If he tried to look downward a spasm of the elevators occurred and the eyes were raised. No explanation was offered.

W. E. Bruner[39]: The chief symptoms were right hemiparesis and impaired vision. The movements of the eyes were perfectly normal in all directions except upward, in which direction there was scarcely any movement at all, not more than five degrees, but there was no diplopia. Slight nystagmus was observed when the patient attempted to look upward. No necropsy.

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[35] Verrey. Revue Médicale de la Suisse Romande., 1893, Vol. 13, p. 220.

[36] Sharkey. Brain, Vol. 17, 1894, p. 238.

[37] Sauvineau. Receuil d'Ophtalmologie, 1894, p. 592.

[38] Cited by Teillais. Annales d'Oculistique, Vol. 122, 1899, p. 19.

[39] Bruner. Columbus Med. Journ., Vol. 14, 1895, p. 505.

Hoesslin[40]: A boy, 9 years old, had had increased thirst and polyuria, headache, vomiting, ataxia, loss of muscular power in the lower limbs, and pupils dilated *ad maximum*. The eyes could be moved to the left, right and downward, but upward movement above the horizontal was impossible. He had bilateral choked discs. Both superior recti, both inferior oblique and the sphincter pupillæ were paralyzed. A spindle cell sarcoma was found growing from the pineal gland and corpora quadrigemina.

C. K. Mills[41]: The patient was unable to raise his eyes beyond the horizontal plane. The left eye diverged slightly, but there was no true lateral paralysis. The pupils were equal, reacting to light and in accommodation, both individually and consensually, but their movements were sluggish. The right field was concentrically limited to a decided degree, and slight concentric limitation was present on the left. The media and fundi were healthy. The central acuity of vision was 20-40 in the right eye and 20-30 in the left. Examination showed difficulty in speaking. The tongue was protruded slightly and a little to the left, and the patient showed some tendency to drooping. He complained of a general feeling of weakness in both legs. Both knee-jerks were exaggerated and a slight, probably spurious, ankle clonus was present on the left side.

Nothnagel[42]: Case 3, as given by Bach, there was bilateral ptosis. The movements of the eyes, especially of the left, were much impaired outwards and upwards. A tumor was found in the third ventricle, and the aqueduct of Sylvius was displaced. Pressure was probable upon the ocular nuclei and root fibers.

Basevi[42]: As given by Bach, associated paralyses were present (oblique superior and inferior rectus muscles, with disturbance of convergence). The corpora quadrigemina were scarcely recognizable.

Teillais[43]: There was complete paralysis of upward and downward movement of the eyeballs, with integrity of lateral movements, also complete paralysis of convergence, and yet the internal rectus of each side showed no impairment in lateral movements. The irides reacted to light, and the pupils were equal. There was no lesion of the fundus, no amblyopia, and the visual fields were normal. This condition lasted two months.

The man had syphilis and diabetes. The symptoms began in an apoplectic attack, and he remained almost unconscious a month without paralysis of the limbs. Only during the first day of his attack had he any difficulty in raising the eyelids. He had very few signs, other than the ocular, of involvement of his nervous system. He was very drowsy and slept much, and his intelligence was affected. He had dysarthria, and the left upper limb was used in preference to the right, and the tendon reflexes on the right side were prompter. No necropsy was obtained in this case.

Poulard[44]: The patient had an apoplectic attack and loss of consciousness during six hours. Following this she had diplopia. Upward movement, and movements to right and left were normal, but associated downward movement was almost entirely lost; the pupils did not pass below the horizontal, and the patient was unable to follow an object downward. Convergence was much affected. There was some insufficiency of the left internal rectus, and also insufficiency in elevation of the left eye, although it was impossible to say whether the superior rectus or inferior oblique was at fault. In going down stairs the woman flexed the head *ad maximum*. The iritic reflex to light and visual acuity and visual fields were normal. Nervous symptoms, other than ocular, were slightly marked, the legs were a little stiff at times, and the tendon reflexes were exaggerated. There was no necropsy in the case.

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[40] Hoesslin. Münch med. Wochenschrift, March 31, 1896, p. 292.
[41] Mills. "The Nervous System and Its Diseases," 1898, p. 833.
[42] Bach. Zeitschrift für Augenheilkunde, Vol. 1, 1899, pp. 315, 455.
[43] Teillais. Annales d'Oculistique, Vol. 122, p. 19.
[44] Poulard. Revue Neurologique, 1901, p. 158.

Gordinier[45]: The patient was a man, aged 21 years. He had double optic neuritis, passing on to atrophy, intense and continuous headache, vomiting, dizziness, slow cerebration and gradual loss of memory, internal ophthalmoplegia, with double incomplete external ophthalmoplegia, marked cerebellar gait, coarse tremor of the hands, ataxia in the left leg, and choreiform movements. It is stated that there was slight convergence of eyeballs, and that the movements of the eyeballs were normal except upward and downward movements, both of which were practically abolished, and that there was slight double ptosis.

A neuroglioma, measuring 4x3x2½ cm., was found projecting from the superior worm of the cerebellum. It had involved by actual ingrowth the posterior part of the corpora quadrigemina on each side, and had destroyed almost completely the interior of the right posterior colliculus, leaving but a superficial shell of cortex; the left posterior colliculus being similarly, although much less, involved. The anterior colliculi did not appear to be affected. The aqueduct of Sylvius was occluded, the third nerve nuclei were distorted, and the left nucleus contained a less number of cells than its fellow of the opposite side. The nucleus of each side contained numerous atrophic cells devoid of processes and without nuclei; other cells had lost their processes and showed much alteration. The dorsal part of the oculomotor nuclei was most affected.

Crouzon, Marie, Babinski[46]: The patient, a man, had an apoplectic attack lasting seventeen hours, and became blind. During the attack he did not have stertorous breathing nor evacuation of the bladder or bowels. The coma disappeared suddenly, but the patient was delirious for several weeks, and did not recognize persons or objects. The speech was slow and hesitating and the visual fields were contracted. The eyeballs were turned upward; when he threw his head as far backward as possible and followed with his eyes a finger slowly lowered the eyeballs moved downward, but if his head were erect and he attempted to look at his feet he bent his head forward and the eyeballs went forcibly backward.

Raymond and Cestan[47]: Case 3. A man, 43 years of age, had left sensori-motor hemiplegia. Eight days after a severe mental shock his speech became affected and he became paralyzed on the left side and had diplopia. The tendon reflexes on the left side were exaggerated and Babinski's sign was obtained. There was no ptosis. The fundus was normal. The pupils were equal, and the reaction to light and in accommodation was normal. The man had paralysis of associated ocular movements both toward the left and toward the right, but more toward the left There was also marked paralysis of upward associated movement. Downward associated movement was almost normal. Convergence was slightly affected. No necropsy was obtained. This condition had existed ten years.

Noguès and Sirol[48]: A woman, 50 years of age, had been in bed about three weeks because she had lost her appetite, had difficulty in sleeping, had lassitude and was unable to work, had chills, and probably fever, with palpitation. She noticed the first day she arose from her bed that she could not look upward. She was found to be unable to look above a certain point a little below the horizontal. Downward and lateral movements and convergence were normal. When the upper lids were lowered she could raise her eyes above the point she could reach if her eyes were open. The fundus was normal. There seemed to be no other signs of organic disease of the nervous system. Because of the absence of an apoplectic seizure and of other symptoms of an organic nature, and of paralysis of convergence, and because of certain hysterical manifestations and of the probably sudden development of the paralysis, Noguès and Sirol regarded the paralysis of upward associated movement as hysterical. It seems to me the hysterical nature of this case has not been established.

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[45] Gordinier. JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 28, 1901, p. 543.

[46] Revue Neurologique, 1901, p. 428.

[47] Raymond and Cestan. Revue Neurologique, 1901, p. 70.

[48] Noguès and Sirol. Revue Neurologique, 1901, p. 290.

Kornilow[49]: Case 1. A child, 6 years old, had complete paralysis in upward and downward movement, some impairment of convergence, and slight slowness in the reaction in accommodation and light. Movements of the eyeballs laterally were normal. Recovery in this case was almost complete.

Case 2. A child, 4 years old, had slight bilateral ptosis, and the left eyeball was turned somewhat inward. Movement of the eyeballs toward the right was almost normal, downward entirely normal, but toward the left the movement was much impaired, especially in the left eye, and upward associated movement was almost impossible. Convergence was normal.

The first case was supposed to be one of poliencephalitis superior of Wernicke; the second, one of tubercle.

In the first case there was slight weakness of the lower part of the right facial nerve supply and of the right hypoglossus; in the second, paresis of the right facial nerve and of the right upper and lower limbs. Ataxia was present in both cases.

Posey[50]: Case 1. Given in detail by me as Case 4 (Frank S.) of my report.

Case 2. The patient, a woman, was 65 years of age. She had had an attack of apoplexy and was hemiplegic on the right side. Neither eye could be rotated above the horizontal plane, though the other movements, including convergence, were preserved. The reaction of the irides to light and in accommodation was sluggish. The pupils were nearly equal, 3 mm. and 2½ mm., respectively. The fundi were normal. Corrected vision equaled 5-6 in each eye. There was no hemianopsia. The condition was unchanged after two years.

J. Porter Parkinson[51]: A girl, 11 years of age, began to stagger when 7 years of age. Sight failed. Headache did not occur. Movements of hands were incoördinate. The deep reflexes were increased. Hearing in the left ear was slightly impaired. Reaction of pupils was sluggish to light and in accommodation. The upward and lateral movements of the eyes were limited, especially the former. Discs were pale, but there was no evidence of optic neuritis. Speech was slow and hesitating. Necropsy was not obtained.

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[49] Kornilow. Deutsche Zeitschrift für Nervenheilkunde, Vol. 23, 1903. p. 417.

[50] Posey. Annals of Ophthalmology, July, 1904.

[51] Parkinson. The British Journal of Children's Diseases, Jan., 1905, p. 23.

Note: Two cases of paralysis of associated upward and downward movements, but not of associated lateral movements, have been reported by Gruner and Bertolotti too late to be included in this paper (*Nouvelle Iconographie de la Salpêtrière*, 1905, No. 2, p. 159).. In one case a tumor of the tegmentum of the cerebral peduncles in the region of the corpora quadrigemina was supposed to be present; in the other a tubercle had destroyed the oculomotor nuclei. A clinical case by W. A. Turner (*Brain*, 1898, Vol. 21, p. 341) also should be included.

TABLE SHOWING CASES OF PARALYSIS OF ASSOCIATED UPWARD OR DOWNWARD MOVEMENT.

	Paralysis	Pupils	Fundus	Lateral Movements	Ptosis	Convergence	Result of Lesion.	Other Symptoms,	Third nuclei.
Henoch 1864	Upward movement	Dilated Reaction slow		Normal Later deviation of right eye inward Eyes turned downward			Tub. meningitis and tubercle of l. post. part of corp. quad.	Vomiting, drowsiness, paresis of r. arm, and later, of both r. limbs and r. side of face	Condition not stated.
Wernicke 1876	Upward and downward		No changes	Normal	None		Apoplectic focus in r. optic thal., len. nuc. and ant. part r. side corp. quad.	Apoplexy L. hemiplegia	Involved on r. side
Priestley Smith 1876 Case 4	Upward			Normal Later imperfect Downward normal			Result not stated	Headache, vomiting, vertigo, diplopia, constant convergence of eyes	
Priestley Smith 1876 Case 5	Upward			One or the other eye deviated inward Outward, inward and downward nearly or entirely normal			Ocular symptoms disappeared	Headache, vertigo, temporary unconsciousness	
Nieden 1880	Upward		No fundus changes	Downward, outward and inward normal			Recovery complete, lesion supposed to be hemorrhage	Attempts to look upward caused eyes to be drawn backward and forward Vision disturbed	
Gowers 1880-'81	Upward	Acted very little to light, and not at all to accom.	Double optic neuritis	All directions free but upward			Small tumor in middle line behind post. quad. bodies, damaging them slightly	Headache, vomiting, weakness of legs, convulsions, patellar reflexes exaggerated	

TABLE SHOWING CASES OF PARALYSIS OF ASSOCIATED UPWARD OR DOWNWARD MOVEMENT.

Parinaud 1883 Case 4	Upward and downward	Moderately contracted and a little unequal Reaction to light lost		Normal	None	Lost	No necropsy	Polyuria, heaviness of head, difficulty in standing, vision disturbed
Parinaud 1883 Case 5	Upward		No lesions	Normal, as also downward		Lost		Headache, diplopia, numbness of lower limbs, followed by weakness
Ormerod 1884	Upward		No definite neuritis	Other ocular movements normal				Nystagmus looking upward Had a similar attack 15 years previously
Hope 1888	Upward, downward much impaired	Rigidity of pupil	Slight choked discs	Lateral movements normal		Said by Bach to be preserved	Sarcoma in the corp. quad. aqueduct preserved only in post. part.	Headache, gen. tremor, r. deafness, vomiting
Reinhold 1886	Upward		No optic neuritis		Bilateral		Gliosarcoma of pineal gland Corp. quad and aque. compressed	Vertigo, headache, paresis of each ex. rectus, vomiting, disturbed vision, nystagmus attempting to look upward
Thomsen 1887	Upward	Small and unequal Reaction to light affected	Pallor of discs	Right, left and downward normal, but with nystagmus			Gumma involving intramedullary 3d nerves	Failure of memory and intelligence, headache, vertigo, weakness, tremor, rigidity of limbs

Nothnagel 1889	Upward and downward		Choked discs	Lateral movements except outward movement of the left eye seem to have been good				Headache, diplopia, paresthesia of left limbs, epileptic attacks, paresis of left limbs, ataxia of left upper limb	
Gee 1890	Downward		Discs white, margin blurred vessels small, atrophy following neuritis		None		Sarcoma involving corp. quad.	Vomiting, headache, ataxia, convulsions, weakness of limbs, retraction of head. Both eyes turned upward, vertical nystagmus	
Eisenlohr 1890	Upward and downward	R. pupil dilated Reaction slow	Choked discs		Right ptosis		Bullet in right side corp. quad.	Drowsiness, vertigo, tremor of l. arm, polyuria, headache, vomiting, stupor, ataxia	A portion 3d nuc. injured
Lichtheim 1892 Case 1	Upward	Rigidity of pupils	Optic atrophy	One 6th paralyzed			Glioma of corp. quad. extending into r. optic thal.	Signs of brain tumor	
Lichtheim Case 2	Upward		Choked discs	One 6th paralyzed			Tumor in corp. quad and optic thal.	Vertigo, ringing in ears, ataxia	
Lichtheim 1892 Case 3	Upward		Optic neuritis	Left 6 paralyzed			Caseous tumor in corp. quad.	Headache, diarrhea, vomiting, weakness of r. side of body	

TABLE SHOWING CASES OF PARALYSIS OF ASSOCIATED UPWARD OR DOWNWARD MOVEMENT.

Parinaud 1892	Upward	Normal size Reaction to light and con. normal	No lesion	Lateral movements imperfect. Downward good		Normal	No necropsy	Paralysis of left face and left upper limb (of short duration) Partial l. homonymous hemianopsia
Verrey 1893	Upward and downward						Recovery	Tendency to go to the left
Sharkey 1894	Upward, downward defective	Reaction to light preserved, and accom. but sluggish	Optic neuritis	Lat. movements good		Converg. defective	Tumor of corp. quad and cerebral ped.	Right deafness, numbness r. half body, r. hemianopsia, sensation r. half body disturbed, speech thick, nystagmus, loss of power r. side
Sauvineau 1894	Upward, downward feeble	Pupils equal, of normal size Reaction in accom. normal L. reflex affected	No fundus changes	Lat. normal, but nystagmus	No ptosis	Conv. lost	No necropsy	Apoplectic attack, transitory r. hemiparesis, diplopia
Schroeder 1894 Cited by Bach and Teillais	Downward							Fever, headache, vomiting, convulsions, at first complete ophthalmoplegia Attempt to look downward caused spasm of elevators

Bruner 1895	Upward			Lat. and downward normal				R. hemiparesis, impaired vision, slight nystagmus attempting to look upward
Hoesslin 1896	Upward	Pupils much dilated	Choked discs	Lat. and downward good			Sarcoma growing fr. pineal gland and corp. quad.	Increased thirst, polyuria, headache, vomiting, ataxia, loss of power in lower limbs
Mills 1898	Upward	Pupils equal, reacted to light and accom, but sluggishly	Fundus normal				No necropsy	Slight difficulty in speaking, tongue protruded to left, weakness of legs
Nothnagel Cited by Bach	Upward and outward				Bilat. ptosis		Tumor of 3d ven. aque. displaced. Pressure prob. on 3d neuclei	
Basevi Cited by Bach 1899	Downward					Con. affected	Lesion of corp. quad.	
Teillais 1899	Upward and downward	Reaction to light preserved	No fundus changes	Lat. normal	Ptosis 1 day	Con. lost	No necropsy	Apoplexy, unconsciousness, drowsiness, impaired intellect, dysarthria
Poulard 1901	Downward	Reaction to light normal		Upward and lat. normal L. int. rectus affected		Con. impaired	No necropsy	Apoplexy, diplopia

TABLE SHOWING CASES OF PARALYSIS OF ASSOCIATED UPWARD OR DOWNWARD MOVEMENT.

Gordinier 1901	Upward and downward	Internal ophthalmoplegia	Optic neuritis	Lat. normal	Double ptosis	Con. slight	Neuroglioma invading post. part. corp. quad. Aq. occluded	Headache, vomiting, dizziness, impaired mentality, cerebellar gait, tremor	Third nuclei distorted
Cruzon Babinski Marie Parinaud 1901	Downward							Apoplexy, delirium, slow speech	
Raymond and Cestan 1901	Upward and lat., both r. and l.	Pupils equal Reaction to light and accom. normal	Fundus normal	Downward almost normal	No ptosis	Con. little affected	No necropsy	Left sensori-motor hemiplegia, affected speech, diplopia, exag. tendon reflexes, Babinski	
Noguès Sirol 1901	Upward		Fundus normal	Downward and lat. normal		Con. normal		Insomnia, lassitude	
Kornilow 1903 Case 1	Upward and downward	Reaction to light and accom. slow		Lat normal		Con. impaired	Recovery	Weakness right 7 and 12, ataxia	
Kornilow 1903 Case 2	Upward and left lateral			Left eye turned inward, r. almost normal, down normal, but to left impaired	Bilat. ptosis	Con. normal		Paresis right 7, right upper and lower limbs, ataxia	
Posey Case 1 is given below by me as Case 4, Sherry									

Case 2 1904	Upward movement	Reaction slow	Fundus normal	Good	None	Preserved		Apoplexy and right hemiplegia	
Parkinson 1905	Upward	Slow to light and accommodation	Discs pale but no optic neuritis	Lat. movements limited				Ataxia, exaggerated reflexes, slight impairment of hearing in left ear, speech slow and hesitating	

ORIGINAL CASES

Case 1 Galik	Upward	Reaction good	Optic neuritis	Lat. movements impaired	None	Preserved	Tubercle on left side of pons	Right hemiplegia of limbs, left palsy of face, involvement of each ext. rectus and left palsy of masseter, exaggeration of right tendon reflexes, hearing affected on left side, left neuroparalytic keratitis	3d nuclei not involved
Case 2 Moore	Upward	Prompt in each eye		Nystagmus in lateral movements Paralysis of movement of both eyeballs to right and paresis of both to the left	Slight ptosis	Preserved but impaired	Glioma of right side of pons	Weakness of each 7, left paresis of tongue, right paralysis of muscles of mastication, paralysis of left limbs, bulbar speech	3d nucleus much degenerated
Case 3 F. D.	Upward downward and lateral	Reaction prompt to light		Inward deviation of right eye	None	Preserved tho' not normal	Hemorrhage in r. tegmentum of pons	Paralysis of r. side of face, of r. side of tongue and of r. muscles of mastication Paresis of left limbs	

TABLE SHOWING CASES OF PARALYSIS OF ASSOCIATED UPWARD OR DOWNWARD MOVEMENT.

Case 4 Sherry (Reported by Dr. Posey)	At first only upward, later also lateral movements and downward impaired	Reaction prompt to light	Normal, at least in early stage of disease	Later conjugate lateral movements much impaired	None	Lost	Living	Impaired mentality, ataxia of limbs, some exaggeration of tendon reflexes, tendency to fall backward when he sits down
Case 5 A. S. (Reported by Dr Zentmayer)	Upward	Right reaction to light good, left sluggish, also good in accom.	Incipient neuritis(?)	Good, but nystagmic	None	Weak	Recovery with paresis of only left superior rectus, later recovery of this muscle	Dizziness, headache, no ataxia, glycosuria
Case 6 S. H.	Upward	Reaction present not very prompt to light Pupils equal		No impairment	None	Preserved	Living	Impairment of hearing, some general weakness, ankle clonus on each side, especially on the right
Case 7 Roberts	Upward and downward		Choked discs	No impairment	No ptosis	Preserved		Headache
Case 8 Mrs. McM.	Upward	Reaction prompt		Left ext. rectus weak(?); no paralysis of associated lat. movement.	Bilateral	Lost	Recovery twice, with relapses	Convulsions, headache, nausea, vomiting, dizziness, ataxia
Case 9 W. B.	Upward and downward	Unequal, reaction feeble in right eye	Veiling of right disc	Nearly normal	None	Lost	Almost all the ocular movements were restored except the downward movement of right eye	Right hemiplegia, headache

THE BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

February 16, 1905.

The President, Dr. Morton Prince, in the Chair.

Dr. Barrett reported a case of Pituitary Tumor.

Tumor of Pituitary Body.—This was reported by Dr. Miller. Patient, female, about 75 years of age, began to show a memory defect, disorientation, vague delusions of persecution and fear; operation for cataract in 1900; complete amaurosis since that time; no pain in head; no vomiting; progressive mental enfeeblement so that at time of admission (July, 1903) to Taunton Insane Hospital she was deeply demented; still had episodes of fear, thinking she was going to be killed and poisoned.

On admission she weighed but 81 pounds; was too feeble to walk, but had no paralysis; markedly senile habitus with arcus senilis; completely blind; corneal opacities prevented an examination of the eye grounds; no overgrowth of bones or other symptoms of acromegaly; slight cervicodorsal kyphosis; very marked general tremor of all the extremities and of tongue; knee-jerks could not be obtained; no symptoms of increased intracranial pressure; no change in thyroid; enlarged left heart; aortic murmur; dry skin and wrinkled; no falling out of hair; slight trace of albumin in urine. On the psychic side she made the impression of one in the terminal stage of senile dementia. Her death was caused in October, 1904, by the lighting up of an old tubercular process, resulting in tubercular broncho-pneumonia.

The cerebrum, beside the tumor of the hypophysis, showed an area of softening in the left frontal lobe, but there was very little atrophy of the convolutions. The vascular walls did not show any pronounced artereosclerosis. Dr. Miller considered the mental symptoms as the result of the organic changes, and not directly due to the hypophyseal hypertrophy.

The tumor, as shown by the photograph and the specimen, is confined to the pituitary, evidently involving only the anterior glandular portion. It is roughly circular in shape, 22 mm. in largest diameter, somewhat nodular, pale, of firm consistence, with no macroscopic cysts. The optic nerves are strongly adherent to the upper lateral surface; the left optic very much atrophied, the right apparently normal.

The histological nature of the growth is somewhat questionable, but it is closely allied to the adenomata with considerable production of fibrous tissue and an increase of the colloid cysts. It could not be asserted that there is an increase of chromophile cells.

It is, therefore, a condition spoken of as pituitary struma or goiter, a hypertrophy which Boyce and Beadles so fully described in their treatise on pituitary tumors.

Dr. Knapp had reported a case of tumor of the hypophysis some years ago, where a sarcoma lay on the sella turcica, compressing the chiasm. Unfortunately, it was not determined whether the growth started from the glandular portion of the hypophysis or the infundibulum. There were no indications of acromegaly, but there were fairly well marked mental symptoms. Schuster, in his recent study of the mental disturbances of brain tumors, has found such disturbances in 66 per cent. of tumors of the hypophysis; more frequently than in tumors of any other region except in tumors of the corpus callosum and the frontal lobe. The sections of the cord in Dr. Barrett's case show changes in the so-called *Zwischenzone* of Bechkren—the region between the columns of Goll and Burdach—in the vicinity of the third cervical segment. Reichardt, from a study of 35 cases of general paralysis, has shown that in the cases of Argyll-Robertson pupils there was degeneration of this zone at that segment.

Dr. Walton said that the absent reflexes in this case are of interest in connection with the question of the location of the reflex mechanism. He noticed that Batten and Collier found prevailing absence of knee-jerk in cases of cerebral tumor accompanied by this cord degeneration, and it seems not impossible that the injury of afferent tracts of conduction may play a part in this loss. In any event the observation tends to corroborate Grasset's view of the cerebral rather than lumbar location of the dominant centers presiding over the reflexes.

The distribution in Dr. Barrett's case precludes the supposition that the injury is due to stretching of the cervical nerve roots, for the columns of Goll suffered at a lower level than the middle root zone.

A Case of Somnolentia, or Sleep Drunkenness.—This case was reported by Dr. E. W. Taylor. The patient was a strong, non-neurotic man of 31, who for several years had been annoyed by a certain dream state in which he left his bed and performed various purposeless acts, impelled by an impulse of fear over which he had no control. On such occasions this had happened from one to several times a year, he would find himself at a distance from his room, having reached his position on one occasion by going through the window and down to the ground, three stories below, by clinging to the copper water leader. He had had many such experiences. The point of special interest lies in the fact that he remembers perfectly the details of what happens between the time of leaving his bed and his final awakening some time after. He wakes from the condition suddenly and goes back to bed without nervous perturbation, and again quickly falls asleep. The condition, as suggested by Dana, might have important medico-legal bearings, and it is also of interest as indicating a curious modification of ordinary sleep-walking in a person apparently far removed from hysteria.

Dr. Hall said that in Dr. Taylor's case we have an interesting problem presented: How closely related are the states of consciousness in somnolentia and hysteria. Janet emphasizes the splitting-up, complexion division of conscious states as an explanation of hysterical manifestations. On a higher plane may we not have an analogous instability in states of consciousness in many sleep-walking cases? In the present instance we have a definite phobia with an urgent obsessional demand which is followed out—a systematized state of consciousness, if you please—but at the same time marked by a dominant, if not, indeed, a fulminating excess in active attention, "the interaction between self and circumstance . . . experience" being in abeyance. Ordinary sensory stimuli at the onset fail to arouse normal consciousness; later, impressions far more feeble disturb sequence of thought and action, and the patient suddenly returns to a full comprehension of himself as related to surroundings. Dr. Taylor remarks upon the absence of the physical stigmata of hysteria. Have we not in the case presented, at least a psychic equivalent, or a phase, at least, of that interesting disorder?

Some Facts Regarding the Early Care of the Insane in Massachusetts, with Special Reference to the Boston Almshouse.—This paper was read by Dr. Ayer.

How well prepared the colonists were to put into practice what they had been learning—the Pilgrims, from residence in a university town and not far from Utrecht, where stood the only asylum for the insane then in Northern Europe; both Pilgrims and Puritans fresh from the discussion of the Elizabethan Poor-Laws of 1601—is evident by their charitable work under new conditions, and by conscientiously reasoning out all medical problems which came before them, Winthrop and Roger Williams being especially considerate and thoughtful. If they were sometimes harsh in their decisions, it was due to incorrect reasoning.

The laws on insanity during the first two centuries; the contrast between the few laws on insanity before 1816 and the classified laws upon our statute book to-day was shown by photographic copies of the order of the General Court of the Colony:

(*a*) 1676, intrusting to the Selectmen the care of "Distracted, and of their estates in the time of their distemperature."

(*b*) 1693-4 (Province Act), an Act for the "Reliefe of Ideots and Distracted Persons" giving the care to Justices and Selectmen, or Overseers of Poor; "Acts in Addition," 1708-9 and 1726-7, appointing guardians; 1731-2, 1737-8 and 1776-7, including the incapacitated deaf and dumb, and also appointing guardians for children of idiots; summed up (for most part), after the Revolution, in an "Act empowering Judges of Probate to appoint guardians to minors and others." (Mass. State Law, March 10, 1784, Sections 3, 4, 5, 6, and 7 part.) And

(*c*) Section 3 of an "Act for Suppressing Rogues, Vagabonds, Common Beggars, and other Idle, Disorderly and Lewd Persons" (1798), allowing Justices to confine lunatics, dangerously mad, in the House of Correction.

Reports of the Selectmen upon the Judge of Probates' warrant were read, showing much thought and consideration on the part of the Selectmen, *e. g.:*

"Pursuant to direction, we have made strict enquiry into the character and behavior of H——— D———, and find that "altho at some times he has lucid intervals, yet at other times he is so wild and ungovernable that we are of opinion he is nowise capable of the management of himself or his own affairs." Signed by five Selectmen, Boston. Aug. 20, 1736.

In 1771 the Selectmen say of James Otis, Esq., that they are "fully of opinion that he is a person *non compos mentis,* and incapable of taking care of his Person and effects."

In 1777: "Said Otis is in general in such a state of mind as renders him incapable to take care of himself or his affairs.

The Almshouse, on Beacon Street, built from legacies and gifts about 1662, was a credit to a town which did much for its sick and poor. It preceded the Almshouse in Philadelphia (1731), from which Blockley and Philadelphia Hospital trace their descent; as well as the New York Almshouse (1736), from which Bellevue descended.

We know almost nothing of its early history, but in 1682, having been burnt, it was voted by the town to rebuild. Donations were not sufficient and a "rate" had to be made for the remaining £329 unpaid. This is discussed in Vol. 1 of Boston Town Records.

The Almshouse, L-shaped, was supplemented by the Bridewell, built in 1720 for the dissolute and insane. In front of the Bridewell, and running down Sentry (now Park) Street, followed the long and narrow workhouse built in 1739. The Almshouse was continued ninety feet to the present corner of Park and Beacon Streets in 1741, and the fence running down to the Granary (brought to Park Street corner in 1729) inclosed a very attractive yard, with the Granary Burying Ground as a background. The history was gone into in some slight detail. All the great events of New England of an historic period can be read between the lines of the scanty records.

. . . Frequent attempts were made to keep the worthy poor by themselves, for it was always tending to become a "Catch-all for Ideots, Epileptics. Incurables, Incompetents, Aged, Abandoned Children, Women for Confinement, considerable number of Insane, Deaf, Blind, Dumb, all Dumped together."

It was described how in 1746, the Selectmen inquired about "Bridewell House adjoining the Work House, and the conveniency thereof for the Reception of Distracted persons," and the wish was expressed that the physicians of the time had then established the first Insane Hospital in America.

Again, in 1764, there was another lost opportunity, when Thomas Hancock died leaving £600 for an Insane Hospital. The town at first voted to accept it and name it the "Hancock," but in 1767 the plan fell through, the Province not being willing to add to the donation. The Town Records, as we have them, end abruptly. Perhaps the town feared that the Province would not repay satisfactorily for the care of the Province poor. The town had not been recompensed satisfactorily by the Province in times past for Almshouse inmates who had "crept in," and the town now felt too poor to bear the expense of outside patients—this was the explanation which suggested itself to the writer.

The Almshouse suffered during the Revolution, and later became much criticised as ill-adapted to its purpose. In December, 1800, a change was made from the old buildings on Park Street to the pretentious new building on Leverett Street. Vol. 33 of Boston Town Records will soon appear, and will, probably, show the same trouble with grouping defective classes with the sick and insane which we have described during the previous century in the combination of Almshouse, Bridewell and Workhouse.

One statement can be made in favor of the new Almshouse. It was fortunate in medical attendants—Dr. James Jackson and Dr. John C. Warren. Bowditch's History of the Massachusetts General Hospital quotes from a circular dated Aug. 20, 1810, signed by Drs. Jackson and Warren, in behalf of a new hospital, the following: "It is very desirable to a number of respectable gentlemen that a hospital for the reception of lunatics and other sick persons should be established in this town."

Our Leverett Street Almshouse moved to South Boston in 1825, but the Boston Insane Hospital did not grow out of it until 1839.

Periscope

NOUVELLE INCONGRAPHIE DE LA SALPETRIERE

(No. 6, Nov.-Dec., 1904.)

1. Disturbances of Reading, Writing and of Speech in Dementia Paralytica. **Joffroy.**
2. A Case of Acute Anterior Poliomyelitis of an Adult, with Medullary Lesions Focally Distributed. **Leri.** S. A. K. **Wilson.**
3. Myotonia with Muscular Atrophy. **Lannois.**
4. Vasomotor Symptoms in Hysteria. **Genevrier.**
5. Trophic Ulcerations in Two Cases of Catatonic Dementia. **Trepsat.**
6. Physical, Physiological and Psychical Stigmata of Degeneration in Animals, Particularly in the Horse; A Clinical Study. **Rudler Chomel.**
7. The Dentist Picture of Andrea Cefaly. **Bianchini.**
8. The Miracle of the Blind Cured; After a Dutch Tapestry of the Sixteenth Century. A. **Marie.**
9. Dropsy in Art. **Meige.**

1. *Reading, Writing, in Dementia Paralytica.*—The author, who has devoted a great deal of time to this subject, divides the symptoms which have to do with written words into two divisions, that of caligraphic and that of psychographic. The former refers more to the external formation of the letters, and the latter to the psychical side. Speech is likewise divided into these two classes of disturbances, those having to do with the mechanical side and those which are concerned with the degree of intelligence displayed in the speech. To the former the name arthrolalia and to the latter psycholalia is given. Examples are given of these disturbances, with copies of the handwriting in the case of the disturbances in writing.

2. *Anterior Poliomyelitis.*—The existence of an acute anterior poliomyelitis in the adult, with the localization of the process similar to that of the infant, has been the subject of much discussion. Dejerine and Thomas remark that, although the disease must be admitted as a possibility, yet no autopsy has as yet been made which determined the existence of the condition. This paper describes a case of a man 28 years old, who presented all the symptoms of the disease as it is met with in the adult, and in the cord there were found foci located in the lumbar and the cervical enlargements in which the same appearances exactly as met with in the infant were found. There were areas of degeneration of an infectious origin. There was a double process in the cord which destroyed the anterior horns in the lumbar and in the cervical enlargements. In both regions the left side was more affected than the right. This corresponded to the clinical appearance.

3. *Myotonia and Muscular Atrophy.*—The author of this paper gives the title myopathy with hypertonia, or Thomsen's disease with muscular atrophy, as describing the condition which he presents. The case is a rare one, and serves the purpose of bringing into relation the primary myopathies, and Thomsen's disease, and serves also as an argument for the myopathic nature of the latter. The patient, a man aged 37 years, is the subject of the following symptoms: Muscular atrophy, segmental in distribution, occupying the lower arms and legs. Commenced at four years with pain and paresis. Loss of knee-jerk. Myotonia in the form as in Thomsen's disease. The reports of such cases are of interest, because they serve to clear up the confusion which various conditions of these mus-

cular diseases have produced. There is evidently a close connection between myotonia and myopathy. This case seems to suggest that Thomsen's disease is myopathic in nature.

4. *Vasomotor Disturbances in Hysteria.*—A case of hysteria which presented various symptoms due to peripheral disturbances of the circulation, such as edema, cyanosis and syncope of the extremities, localized sweating, etc. The trophic disturbances in hysteria have given rise to much discussion, but it seems pretty well agreed at present that they are due to vascular spasm.

5. *Trophisms in Catatonia.*—Two cases of catatonia which show lesions due to trophic disturbances. Case 1. Dementia præcox with catatonia. Stupor, negativismus, paradoxical attitudes. Pseudo-edema, and generalized pemphigus. Case 2. Dementia præcox, absolute stupor, with violent impulsive acts. Suggestibility. Pseudo-edema with scarring.

(No. 1, Jan.-Feb., 1905.)

1. Friedreich's Disease and Hereditary Cerebellar Aatxia. F. RAYMOND.
2. Medullary Sclerosis. L. REVILLIOD.
3. Early Multiple Sclerosis, or the Cerebellar Syndrome of Babinski. G. SCHERB.
4. Amnesic Aphasia. A. HALIPRE.
5. The Rôle of the Spinal Muscles in the Normal Gait of Man. H. LAMY.
6. Senile Deformities of the Skeleton Simulating Paget's Disease. P. MOCQUOT and F. MOUTIER.
7. Dementia; Senile Puerilism. E. DUPRÉ.
8. Ambroise Paré. DEBOVE.
9. Two Saints Cured of Madness. MEIGE and RUDLER.
10. Expansion of the Toe in Art. LAIGNEL-LAVASTINE.

1. *Freidreich's Ataxia.*—This is apparently a clinical introduction to a further study of this disease to be presented at another time by the author. He calls attention to his former paper on this subject, as well as to the pathology of two cases studied from his clinic by Philippe and Oberthur. Further, an effort is made to unite under one group all these cases in which the group of symptoms is present. This will be done by the same sort of careful analysis that a zoologist or botanist makes use of in the determination of the various groups in his classification. A case is presented in which the symptoms of the disease are well shown. The author closes his clinical lecture by recalling the fact that the work of the past in this disease from the point of view of pathology, is not reliable, on account of the use of methods that were not sufficiently delicate, and he calls attention also to the fact that the way is open for further investigation into the field.

2. *Medullary Sclerosis.*—The case here described is complicated. There was the following sequence of symptoms: Fracture of the right foot, four months later left hemiparaplegia, hemianesthesia and sensory disturbances of the lower right extremity, crossed thermoanesthesia, the right to cold, the left to heat. Six months after, hematorrhachis, and finally cure. According to the symptoms the three systems involved were: 1. The motor tract of the left lower extremity, pyramidal tract. 2. The grey substance about the central canal. 3. The sensory tract of the right and the thermic tract for heat of the same side. Two sorts of diagnosis were advanced, one anatomical in nature and one pathogenic. The first was a transverse myelitis, the other metatraumatic segmentary myelitis. The author is inclined to think that this forms a special variety of disease which has not been noticed before by neurologists. The great interest in the case lies in the fact that the patient made a good recovery following therapeutic procedures; among which hydrotherapy played the most important part.

3. ***Multiple Sclerosis and Babinski Syndrome.***—This paper contains the photograph of a patient whom the author has studied for five years, with the idea that the diagnosis of multiple sclerosis might sometime be made. The case then presented the following: scanning speech, spasmodic ataxic gait; but there was no intention tremor present, no increase in reflexes and no nystagmus. Under the term cerebellar syndrome Babinski has grouped all the symptoms which may be considered as defects of cerebellar function. This is the asynergia of the cerebellum. These include the asynergia, asthenia, atonia. A case which shows all of these features is described, and the fact that the symptoms have remained stationary for five years is characteristic of the condition. The photographs add much to the understanding of the case.

4. *Amnesic Aphasia.*—Pitres has given the most precise definition of the term "amnesic aphasia": Any patient who comprehends that which one says, who can read out loud, who pronounces and writes easily the words which he remembers and is not prevented from pronouncing or writing the others the memory of which he is not able to recall at the opportune moment, such a patient has pure amnesic aphasia. Case: Woman aged 67 years, attacked with a right-sided hemiplegia. Amnesic aphasia, paraphemia, verbal blindness, incomplete blindness for written words. Paragraphia for spontaneous writing and diction. Autopsy: Softening on the external face of the left hemisphere, starting from left occipital lobe, extending to the superior part of the second parietal fissure, to the first parieto-temporal fissure, to the posterior part of the lobule of the second parietal. Destruction of the subjacent white fibers of the first and second temporal, the cuneus, lingual and the fusiform lobe and the optic radiations of Gratiolet. There is a small focus, very limited in the left paracentral lobule corresponding to the foot of the third right frontal. Pitres notes that the lesion which produces amnesic aphasia does not in every case affect the same location. It may be situated in the left cortex at the level of the parietal or temporal region, in the area or upon the immediate confines of the visual centers or that of the hearing for words. Most often it is located upon the inferior parietal lobule. It does not seem probable that this lobule should be considered as the exclusive center of the function of the memory of words, since it is not always altered when this function is affected.

5. *Spinal Muscles in Gait.*—A careful study of the mechanics of walking, in which the study is confined to the rôle which the spinal muscles play. It is a continuation of the work of Richer along the same lines. These are some of the conclusions noted: 1: The spinal muscles (sacro-lumbar and long dorsal) contract energetically at each step in the normal gait upon a horizontal surface. 2. This contraction is unilateral, and is produced upon the side of the inferior oscillating member. It passes from one side to the other in the same way as the oscillations. 3. It begins at the moment when the toe of the member touches the ground. It is a sudden, quick contraction, produced in the manner of a reflex, which might take place when the foot comes in contact with the ground. It lasts as long as the oscillation of the leg. 4. This contraction has to do with the transference of weight from one leg to the other, and has nothing to do with progression. 5. Its rôle in walking is to assure lateral equilibrium of the trunk.

6. *Senile Deformities.*—A discussion of Paget's disease and the question of the distinction of the senile changes in the skeleton which may simulate the condition. In Paget's disease, in addition to the well-known group of symptoms there is found after death a thickening of the bones of the skeleton. Marie observed that in a certain number of cases in which the symptoms appeared to be similar to those of Paget's disease the bones showed no such alteration; in fact, showed a notable fragility. To this

condition he gave the name of pseudo-Paget. The authors conclude that there are certain deformities of the skeleton depending only upon senile changes, one of the results being an attitude which recalls Paget's disease.

7. *Puerilism.*—Dupré has called attention to a curious condition which he has called puerilism. It is a kind of regression of mentality to that of infancy. It is characterized by a singular modification of thoughts, tastes, tendencies, appetites, language and expression, such a modification as would seem to change the individual to a little infant. This alteration of personality, the pathogenic substratum of which we are in complete ignorance, can be observed in many different pathological states, organic affcions of the brain intoxication and hysteria. The state of puerilism seems to be the clinical expression of the primitive beginnings of personality placed in evidence by the destruction or overturning of the superior planes of our psychical strata. SCHWAB (St. Louis).

REVUE DE PSYCHIATRIE ET DE PSYCHOLOGIE EXPERIMENTALE

(April, 1905.)

1. Insanity in Prisons.—PACTET.
2. Breaking Off of the Superior Alveolar Arch of a Paretic in an Effort at Mastication. HENRI DAMAYE.

1. *Insanity in Prisons.*—The article is a plea for the establishment of a psychiatric service in connection with the prison, based upon a showing that from 5 to 6 per cent. of prisoners have some form of insanity unrecognized, and that about 15 per cent attract attention sufficiently by strange acts to warrant examination.

2. *Fracture of Alveolar Arch.*—An account of a paretic who first tore out the upper incisors and canines by biting his bedding, and finally broke off the portion of the superior alveolar arch corresponding to the insertion of the incisors while masticating his food. WHITE.

JOURNAL DE NEUROLOGIE

(1904, No. 22.)

1. The Relations which Exist Between the Topography of Paralyses and the Alterations of the Motor Centers in Acute Poliomyelitis Anterior. PARHON and J. PAPINIAN.

The authors report the results of microscopical examination of the nervous system in the case of a woman, dead at the age of 82 years, who had had poliomyelitis in early childhood, and discuss the bearing of their findings upon the localization of the muscles of the lower extremity in the cell groups of the anterior horn of the cord. They found extremely atrophied all the muscles of the left leg and foot, also the quadriceps, the adductor magnus and the biceps cruris. The other muscles were normal. In the lumbo-sacral cord the left anterior horn was altogether smaller than the right, and showed a lessened number of cells, distributed as follows: In the first and second lumbar segment the antero-internal and antero-external cell groups were present, but contained considerably fewer cells on the left than on the right. In the third lumbar segment the central and posterior groups were visible, all four groups on the left showing a diminished number of cells. In the lower part of this segment only the antero-external group was well represented, and a similar arrangement was observed in the upper part of the fourth lumbar segment. In the lower part of this segment all the groups were well represented on the right. On the left the antero-internal group alone was present. In the fifth lumbar segment the antero-external, central and postero-external, with a small antero-internal group, were well shown on the right while on the left the postero-external group was entirely wanting. In the first sacral segment not only the postero-external, but also the two central cell groups had disappeared on the left, all the cell groups being well shown on the right, while in the lower part of this segment what he calls the "post-postero-external" group appeared on the right, being absent on the left. In the second sacral segment the same groups were absent on the left, the central group disappearing toward the upper part of the segment. In the third sacral the postero-lateral and post-postero-lateral groups were still absent on the left. Some spinal ganglia from the affected region showed nothing abnormal. Comparing the muscles affected with the cell groups which were missing wholly or in part, the authors conclude, first, that the topography of the paralyses in anterior poliomyelitis is not radicular, but rather medullary, and second, reviewing the findings of a large number of investigators in both clinico-pathological and experimental studies, they conclude that the nuclear localization of the affected muscles in this case agrees very well with that indicated for these muscles by the majority of authors.

(1904, No. 23.)

1. Reflexions on Five Cases of Acute Psychoses Studied Histologically. A. Deroubaix.

In the author's studies, which were confined to the cortex, the methods of Nissl, Heidenhain, Cajal and Marchi were used. Of his five cases three proved to be of general paresis, one was a case of senile dementia with acute exacerbation, and in the fifth the diagnosis of melancholia agitata was made. He does not report anything unusual, but emphasizes the presence in the cases of paresis of perivascular and fibrous changes, and suggests that in the brain we have to do with parenchymatous and interstitial changes, as in other organs.

(1904, No. 24.)

1. Troubles of Psychomotility. D. De Buck.

A discussion of the method of production of psychomotor troubles, as explained on the one hand by the association psychologists and on the other by the apperception school, to which latter the author declares himself an adherent.

(1905, No. 1-2.)

1. Considerations Concerning Dementia Præcox. E. Marandon de Montyel.
2. Some Reflexions upon the Causation of General Paresis in the Department of the Orne. Dr. Coulonjou.
3. Melancholic and Catatonic Stupor. Dr. Deroubaix.

1. *Considerations Concerning Dementia Præcox.*—In a somewhat lengthy and polemic article the author combats the conception of dementia præcox advanced by Kraepelin and his followers, declaring this conception a false one. He objects especially to the name, which implies termination in dementia, whereas these cases are by the Kraepelin adherents found capable of recovery in a proportion variously stated, and reaching as high as 44 per cent. Again, the term precocious strikes him as unsuitable, since the disease is claimed to occur in persons even up to middle life. In the French conception exposed by Christian the term dementia præcox is limited to a dementing psychosis of adolescence, below 25 years, and by this conception the author would stand. He also objects to some of the views of the German school with regard to etiology and symptomatology, finds nervous and insane heredity, exhaustion and trauma to play an important rôle in the causa-

tion of the disease, and regards the symptoms described as entirely reconcilable with those of the primary mental confusion of French authors. A note of national and patriotic feeling seems to run throughout his article, and he closes with the advice to reject thhe "pseudo-dementia præcox" of Kraepelin and to remain "faithful to the old traditions of French psychiatry," which he considers founded on a rock. Doing this will be at the same time "resting upon clinical truth and glorifying our old masters."

2. *Some Reflexions upon the Causation of General Paresis.*—Commenting upon the relative infrequency of general paresis in the Alençon Asylum, which receives its patients from the Department of the Orne—the figure being about 2 per cent. of admissions as compared to 10 to 30 per cent. for other French asylums—the author states that in this region syphilis is as prevalent as elsewhere, while alcoholism is unusually diffused among the population, the lower classes of which are exceptionally ignorant and backward. From these facts he argues that we are, perhaps, inclined to overestimate the importance of syphilis in the etiology of this disease, and to neglect other causes tending to produce exhaustion, particularly of the brain.

3. *Melancholic and Catatonic Stupor.*—The author discusses the psychological causation of stupor in the course of melancholia and in that of catatonia. In the first case there is a "retardation," in the second a "blocking" of impulses. The diagnosis is important as determining the prognosis, which is more favorable in the first condition. He reports two illustrative cases to bring out the points of difference. In the first condition there is shown an extreme slowness and feebleness of response to stimuli, but no active resistance. When, for instance, a limb which has been caused by the examiner to execute certain movements is released it shows no tendency to return to its position. In the second instance, however, there is entire absence of response to stimuli, and such determined resistance to all manipulations that no change in the position of the limb can be effected, combined with obstinate mutism and refusal of food.

(1905, No. 3.)

1. The Psychology of the Sexual Impulsion. N. VASCHIDE and CL. VURPAS.
2. Clonus of the Rectus Abdominus Muscle in Pott's Disease. C. PARHON and J. PAPINIAN.

1. *The Psychology of the Sexual Impulsion.*—The authors, from a series of studies especially on the muscular and blood pressure conditions which accompany sexual excitement, come to the following conclusions: *a.* The genital act consists in a motor tension more or less marked, which at its maximum presents a short tonic stage, followed by a clonic phase, and terminating in a preiod of adynamia or repose. *b.* In the production of this condition the motor image plays the primordial rôle. *c.* In the sexual act the whole motor system is involved. *d.* Erotomania, which is proper to subjects having troubles of the motor image, seems to confirm the importance of the motor image in sexual life. *e.* Motor excitement shows itself equally in circulatory vaso-motor and respiratory phenomena.

The sexual life is due especially to the tendency of the motor image to pass into the motor act.

2. *Clonus of the Rectus Abdominus Muscle in Pott's Disease.*—A description of a case of Potts' disease with spastic paraplegia and great exaggeration of reflexes in the lower extremities in which a clonus of the rectus abdominis could be elicited by firmly stroking with a finger tip across the abdomen in the neighborhood of the umbilicus.

ALLEN (Trenton).

JAHRBUECHER FUR PSYCHIATRIE UND NEUROLOGIE

(Vol. 25, 1904, No. 2, 3.)

1. Multiple Metastatic Carcinoma of the Central Nervous System. O. Fischer.
2. Attempts at Cure of Paralytics. Pilcz.
3. Statistic Study of 206 Cases of Multiple Sclerosis. Berger.
4. Fatigue Pnenomena, Including Those of Vibration Sensation. Neutra.
5. Experiences in the Diagnosis and the Prognosis of the Ménière Symptom-Complex. Fraenkel-Hochwart.

1. *Carcinoma of Central Nervous System.*—This is a study of the central nervous system in a case of metastatic carcinoma. The spinal cord was not examined. The nerve cells in the brain outside of the carcinomatous foci were nowhere to be found in a normal condition, though the changes were limited to slight deviations in the reaction of the Nissl bodies to the stain. The cells in the immediate neighborhood of the carcinomatous nodules as well as those in the nodules themselves showed a great variety of abnormal changes. The blood vessels were much widened, both in the nodules and in the neighboring brain substance. The number of blood vessels in the tumor itself was increased. There were no metastases found in the meninges. The fact that there was found no increase in the volume of the brain is explained on the assumption that the tumor is capable of destroying as much volume of brain substance as it possesses itself. The localization of carcinomatous metastases in the cortex is to be looked upon as the result of an embolic process.

2. *Treatment of Paresis.*—This paper is an account of an interesting therapeutic experiment based upon the well-known fact that some intercurrent febrile affection often exerts a surprising effect upon the course of a chronic mental disease. Sixty-nine cases were given injections of Koch's tuberculin in increasing doses and their course was carefully studied. Data referring to 66 cases of these are at hand. The patients were apparently all suffering from dementia paralytica, though the author leaves this to the inference of the reader. Short clinical histories are given of all the cases so treated. The results are given in a rather vague way, leaving the impression that the treatment was eminently successful, especially in comparison to a certain number of untreated cases which in a measure form a control to these experiments.

3. *Multiple Sclerosis.*—Such a large material of multiple sclerosis deserves the widest attention in respect to the details of the clinical pictures presented. There are 149 men and 66 women in this material. Syphilis was found only three times in the histories. Three per cent. of the cases gave a history of some acute infectious disease. In only 2 cases was an undoubted history of exposure to wet and cold found. In 18 cases there was found a history of trauma antedating the beginning of the symptoms so shortly that there might have been a causal connection between them. Nine per cent. of these cases might have had a traumatic origin. In five cases emotional experiences were given as the cause of the disease. The initial symptom was found to be connected with the lower extremities in 103 cases, generally in some disturbances in gait. General nervousness, headache, vertigo, palpitation, vomiting were found to be the initial symptoms in 19 cases. Disturbances in visual apparatus in 17 cases. A slight dementia was found to be present in 24 cases. Three cases showed evidence of epilepsy. In 3 cases the disease began with apoplectic insult. Difference of the pupil is a very common symptom. Nystagmus is often found in 45 per cent. of this series. In 14 cases the group of symptoms first described by Kuhn in 1896, namely, the tremor accompanying the stationary position of the bulbs, the dissociation of the eye movements and the tremor of the muscles of accommodation were present. Speech dis

turbance was found very frequently; objective anomalies in 56 cases. In 137 cases the gait showed some disturbance, spastic paresis in 37 cases. The sensory disturbances were found seldom, only in 19 cases. In 163 cases the knee-jerks were markedly increased. Foot clonus 123 times. These are only some of the clinical data which this carefully analyzed material shows.

4. *Fatigue Phenomena.*—Should be consulted i noriginal.

5. *Ménière Symptom-Complex.*—This paper is a review of the large material which the author has used in the preparation of his well-known monograph on the subject of the Ménière complex. The material consists of 208 cases, all of which were examined by a competent otologist. The cases have been collected since 1886, so that a revision would give valuable data in respect to prognosis and to the length of time the disease exists. The number of cases in which such information could be obtained is 80. The clasification adopted by the author forms a sufficiently interesting part of his paper of be fully given. Definition: Ménière's Symptom-Complex is a clinical picture consisting of disturbance in hearing and the accompanying trias, tinnitus, vertigo and vomiting, and is often accompanied by headache, cerebellar ataxia, nystagmus, and in rare cases diarrhœa.

I. The condition is found in patients with intact organs of hearing. (1) The apoplectic form (real Ménière's disease of many authors, apoplectic deafness). Anatomical foundation: Hemorrhage into the labyrinth. Infiltration of the acusticus. This happens (a) in cases of normal organs of hearing and in otherwise healthy individuals; (b) in normal organs of hearing, but in people otherwise sick (leucemia nephritis, lues and also in tabes); (2) certain traumatic forms in which the trias followed immediately after the insult (severe concussion, caisson disease, strong detonation).

II. The symptoms occur in ears. the subject of former diseased conditions of acute or chronic character; (a) middle ear diseases; (b) labyrinthine process; (c) processes in outer ear; (d) vertigo symptom in acute and chronic diseases of the acoustic nerve.

III. A result of treatment to the external ear (injections into the ear, catheterization, etc.).

IV. Pseudo-Ménière attacks. Cases are given to illustrate these various conditions, and the points brought out, especially in the way of differential diagnosis, form a valuable contribution to the literature on this subject. SCHWAB (St. Louis).

MISCELLANY

RECURRENT OCULOMOTOR PALSY, WITH A REPORT OF A CASE. William G. Spiller and William Campbell Posey (The American Journal of Medical Sciences, April, 1905).

The patient, a physician, aged 31 years, complained chiefly of diplopia and drooping of right eyelid. On examination there was found to be an almost complete ptosis of the right upper eyelid, and also a paresis of the internal rectus and inferior oblique muscles of the same eye. The left eye was normal, and the pupil of each eye responded normally to light and in accommodation. His family history was negative, except that one sister had attacks of migraine. When the patient was 14 years old he began to have spells of dimness of vision, with flashes of light, and associated with headache, nausea and vomiting. After two years the spectra disappeared, although the attacks of headache and nausea persisted. In January of 1904 he had an attack of pain in the right eye, followed by diplopia, but with no ptosis, from which he recovered completely in about two weeks. Seven months later he had a similar attack, associated with ptosis, at first intermittent, but later permanent and almost complete. This condition lasted almost five months, when the lid raised for a week and then began partially to droop, in which condition it still remains. A complete examination by Dr. Spiller revealed no sign of other disease of the nervous system, such as tabes or paresis, so that the case must be regarded as one of those rare instances of recurrent oculomotor palsy described by Möbius and others. It however differs from those described by Möbius in being only a partial palsy and limited to the external muscles or the eye, and also in the age of the patient, Möbius fixing 25 years as the arbitrary limit in which this symptom-complex occurs. C. D. CAMP.

EXPERIMENTAL EPILEPSY. Zimmern and Dimier (Arch. d'Elect. Med., Oct. 10, 1904).

The authors have shown that intermittent electric currents of low frequency applied to the anterior part of the skull in certain animals can produce most of the symptoms of epilepsy. A gradual increase of the current induces coma, which is maintained as long as the current remains of uniform density. With any irregularity of the current motor phenomena intervene, convulsive movements, as of Jacksonian epilepsy with glottic spasm in the earlier stage, and rise of temperature in the later. With the positive pole the coma is less deep than with the negative. A repetition of the experiment enables the coma to be produced by a much weaker current than in the first instance. A very slight increase of the current beyond that required to produce coma brings about death from paralysis of the respiratory center. It is also observed that death may follow a pulmonary engorgement with extravasation of blood into the respiratory passages and a profuse secretion of mucus. JELLIFFE.

PSYCHIC TRAUMA AND PROGRESSIVE PARALYSIS. H. Kriege (Zeitschrift f. klinische Medicin, LV Naunyn Festschrift).

Kriege describes a case and cites two others from literature in which a severe fright apparently occasioned progressive paralysis. He is sure that vasomotor disturbances may result from intense fright as serious as those from physical concussion of the brain. Syphilis and an inherited predisposition are not excluded in his case. JELLIFFE.

JUVENILE TABES. Giannelli (Riforma Medica, Feb. 25, 1905).

Giannelli reports an interesting case of tabes in a virgin of 17 years who was fairly healthy, with no signs of inherited or acquired syphilis. She went in sea-bathing while menstruating, an experience attended by what she describes as "an intense emotion," and for several days thereafter suffered from pain in her joints. These pains gradually disappeared, however, but after a lapse of three months she had pains of the lancinating type, with severe unilateral headache. These recurred more and more often, and at 22 she displays the typical syndrome of juvenile tabes. The author gives a brief summary of the literature, and calls attention to the fact that a number of cases so recorded belong in the class of Freidreich's ataxia. Some come from a symptom-complex resulting from a specific meningitis, and but few appear as the typical picture of pure tabes.

JELLIFFE.

Book Reviews

MANUAL OF PSYCHIATRY. By J. ROGUES DE FURSAC, M.D., formerly Chief of Clinic at the Medical Faculty of Paris. Authorized Translation from the French, by A. J. Rosanoff, M.D., Junior Assistant Physician, L. I. State Hospital, King's Park, N. Y. Edited by Joseph Collins, M.D., Consulting Neurologist to the Manhattan State Hospital, West. First Edition, John Wiley & Sons, New York.

This little work has been liked so well in France that it has already appeared in French, in a slightly improved form, in a second edition. The translation by Dr. Rosanoff is of the first edition, but will be found useful, as it is based on the new Kraepelin classification, and therefore is, so to speak, up to date. The first part (comprising 120 pages) is devoted to the Etiology, Symptomatology and Practice of Psychiatry; and the second part (220 pages) treats of the individual psychoses, classified as Infectious Psychoses, Psychoses of Exhaustion, Toxic Psychoses, Psychoses of Autointoxication, Psychoses Dependent upon Organic Cerebral Affections, Psychoses of Involution, Psychoses without a Well-Determined Etiology, which are apparently based upon a morbid predisposition; Psychoses Based upon Neuroses and Arrests of Mental Development. Every one who accepted the Kraepelin classification seems to find it necessary to "slightly modify" it for clinical purposes, and it will be noticed that Dr. de Fursac has modified it also to some extent. If I, the reviewer, might offer a criticism, I would say that the author, in his endeavor to condense into a small manual a large subject, has sacrificed detailed descriptions of symptoms of individual psychoses and introduced no illustrative cases. These may not be required for the advanced student, but will be missed by the beginner. ATWOOD (New York).

News and Notes

The Thirteenth Annual Commencement of the Training School for Attendants, State Hospital for the Insane, Danville, Pa., was held on July 13, 1905. The graduates were: Mary Ellen Blue, Sue Beishline, Lillie L. James, Hannah Mary Spargo, Eugene B. Fritz and Albert George Povey.

The National Association for the Study of Epilepsy and the Care and Treatment of Epileptics will hold its next meeting in New York City in November, 1905. It is hoped that all persons who are interested will take part in the November meeting of the National Association in New York. The executive committee wish reports from as many different countries as possible and from each State of the Union. You are invited to report facts, plans, opinions, etc., for the meeting. The transactions of the November meeting and of the three preceding meetings will be published. Please communicate with the secretary as early as possible.

In response to a recent letter many interesting replies have come, but we are anxious to add to the number of such responses. Any fact about this subject will be welcome, either from our own country or from abroad. I earnestly urge you to give this matter a few minutes' attention. Even a few lines will be of interest. All the information collected will be credited to its proper source and the results published in a volume which is designed for general library distribution, and will, no doubt, serve as a book of reference for some years. You are again invited to prepare a paper for the November meeting, or to write a letter to the secretary giving any information about your own or other countries or localities. No item will be too commonplace to be of interest. The very point which is to you trivial and common may be of vital interest in another region. Everett Flood, M.D., Secretary, Palmer, Mass.

WARM WEATHER PROBLEMS

The physician has many difficult ones to face when the heat begins to take hold. But the majority of warm weather cases originate in the digestive organs.

Men and women flood their stomachs with iced drinks, over-eat, over-exert, and eat food which the high temperature has spoiled. The result is rebellion that is often hard to overcome. The summer is the worst time of the year to have a weak stomach, yet that is just when most people have it.

It is important to cut down the food allowance until normal conditions are restored. Yet the patient must be nourished.

Pabst Extract

supplies needed nourishment, in a form readily assimilated. It is the ideal summer food, containing as it does all the ingredients necessary to the human body. Even the patient understands that grain foods are better than meats in warm weather.

And the extract of hops in *Pabst Extract* furnishes a stimulant that assists nature in restoring the digestive organs to normality.

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Vol. 32 September, 1905 No. 9

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Vol. 32 September, 1905 No. 9

THE
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Original Articles

ACUTE ANTERIOR POLIOMYELITIS IN A YOUTH.*

By Theodore A. Hoch, M.D.,
ASSISTANT PHYSICIAN AND PATHOLOGIST, WORCESTER INSANE HOSPITAL, WORCESTER, MASS.

The case to be described is one of sporadic acute anterior poliomyelitis in a boy, 16 years of age, who died 13 weeks after the onset of the disease. Dr. B. T. Burley, of Worcester, observed the case and kindly furnished me with the clinical data.

On August 22nd, 1904, Dr. F. H. Baker called Dr. Burley in consultation to see the following case. H. S., a boy in his 16th year, was a member of a generally healthy family. One sister, however, two years older, had died three months before of tubercular peritonitis; and an older brother had had an ankylosed (tubercular?) knee since childhood. H. S. had always been especially healthy and had grown larger than any of the other children. Five days ago he did not feel as well as usual though he had no pain; felt languid and asked to be excused from doing the chores. The next day he had some headache and alternating hot and cold sensations. The headaches became more severe in the next two days and were accompanied by marked stiffness of the neck. The following night he arose to go to the bathroom and on getting on his feet slumped to the floor. It was found he could not walk and he was carried to his bed. On attempting to pass his urine at this time he failed and he was catheterized by the attending physician. The next morning it was found that the left shoulder could not be raised and the hand used but little. Later in the day the right arm also became weak. At the time of the physician's visit the next day he was told the temperature had been varying from 99.5 to 102.5 degrees, and that there had been at times slight delirium in the last two nights. Three physicians had already seen the boy and the prevailing diagnosis was cerebro-spinal meningitis.

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*Read at a meeting of the New England Psychological Society at Foxborough, Mass., March 28, 1905.

Physical Examination.—A well-developed and well-nourished boy. Face flushed. Skin rather hot and dry. Tongue coated and dry. Pupils equal and react normally to light and distance. Head normal, except for slight retraction and increased tension of the sub-occipital muscles. Heart not enlarged; no murmurs. Pulse 94. Abdomen not abnormal.

Muscular power: Patient could raise neither shoulder. Could use forearm and biceps on right; flexor muscles of forearm on left. Could grasp lightly with both hands; right stronger. Muscles of chest and abdomen not apparently affected, though respiration was not deep. Could raise neither leg from the bed,—in fact all motions of the thighs were lost. Flexion and extension of ankles and toes possible but weak. No paralysis of sphincter ani.

Reflexes: Triceps, quadriceps and Achilles absent. Abdominals and cremasterics lost. Chin and orbicular present. Muscular irritability to mechanical stimuli generally marked.

Sensation: No anesthesia to touch, pain or temperature anywhere. Slight general hyperesthesia, especially of legs, but tenderness was not marked over particular nerves or muscles.

Patient mentally clear. Speech normal. Urination had not been voluntary since the first catheterization. The diagnosis was given as acute anterior poliomyelitis. Prognosis, as to life, good; as to muscular efficiency, poor.

On September 11th the patient was seen again. Within two days after the onset of the paralysis the temperature had dropped to normal, the muscles of the neck had relaxed and the patient began to void urine voluntarily. Since that time there had been slight, gradual improvement in the use of certain muscles of the limbs. He had been on general supportive treatment.

Examination at this time showed that the patient had lost much flesh and that the extremities were somewhat atrophied. Skin, especially of hands, dry and scaly. Reflexes and sensation unchanged from former examination. Heart—systolic soft murmur at base and in neck. Sounds clear at apex. Lungs normal. Patient could now raise the right shoulder slightly and could flex and extend the right arm. Movements of left shoulder, and arm extension, impossible. Flexion of legs lost. Extension weak. Adduction and abduction weak. Outward rotation of right leg possible; of left lost. Movements of toes and ankles practically normal. No toe or wrist drop.

Electrical examination (considerably hampered by failure to use a strong current). To a moderate faradic current all hand and arm muscles contracted except the left triceps. The pectorals and deltoids failed to react to a painful current. The ulnar nerve conduction was normal. Legs—both quadriceps, sartorii and adductors failed to react to a painful current. All muscles below the knees reacted to a fairly strong current. To the galvanic current no muscles showed any increase of irritability and the cathode closure gave a stronger contraction than the anode.

The urine now contained much pus and some blood cells. The patient's appetite was good and his bowels regular. The cystitis was receiving active treatment. Massage was recommended, especially for the extremities.

For two months the patient was not seen again. He had been having massage and, while his strength had but slightly improved, when put in a wheel chair, he had managed to work himself around the room. The cystitis had persisted.

November 11th, 10 p.m. The patient was found reclining in a cushioned wheel chair. Face cyanotic. Rattle in bronchi audible across the room. Accessory respiratory muscles in action. Heart rate 136. Sounds booming; no murmur. Coarse mucous râles all over lungs in front. Bronchial respiration in right axilla and lower front. Heart sounds transmitted loudly through this area. Patient could move the right arm fairly readily and reach above his head. No paralysis of either forearm, though there was considerable weakness. On account of the critical condition of the patient the leg movements were not examined. Oxygen was sent for but before its arrival the dyspnea became extreme. The pulse went to 150 and was irregular. Oxygen administered for three minutes gave very marked relief and within an hour he was resting fairly well in bed, the pulse having dropped to 124. The regular stimulating and supportive treatment for pneumonia was instituted. Next day the whole right chest was flat to percussion, with bronchial respiration. There were coarse râles, numerous on the left. During the night dyspnea and weakness became extreme and the boy died at 5.30 a.m., November 13th, 1904.

The autopsy was performed by Dr. Burley and myself three hours post mortem. The spinal cord only was examined. Body considerably emaciated; muscular atrophy most marked about shoulders and arms, especially the left. Both legs markedly atrophic and about equal. Right chest, front and back, flat to percussion. The spinal cord was removed from the fourth cervical segment downward. Cerebro-spinal fluid clear and moderate in amount. Pial vessels very much injected. Cord somewhat smaller than normal. Consistency fairly firm except in the upper dorsal region, where it was rather soft. Transverse section of the fresh cord in the cervical, dorsal and lumbar region showed slight grayness of the white matter. This was most pronounced in the anterior columns and gradually faded toward the posterior parts of the lateral tracts. The gray matter in every section stood out prominently and was of a light pink color and showed considerable injection and softening in the anterior horn area.

Microscopical Examination.—All of the segments of the cord from the fourth cervical downward were examined. The stains employed were Nissl's methylene blue, hematoxylin and eosin, Weigert's medullated fiber stain, Weigert's neuroglia stain and Marchi's method for degenerated fibers. All of the segments

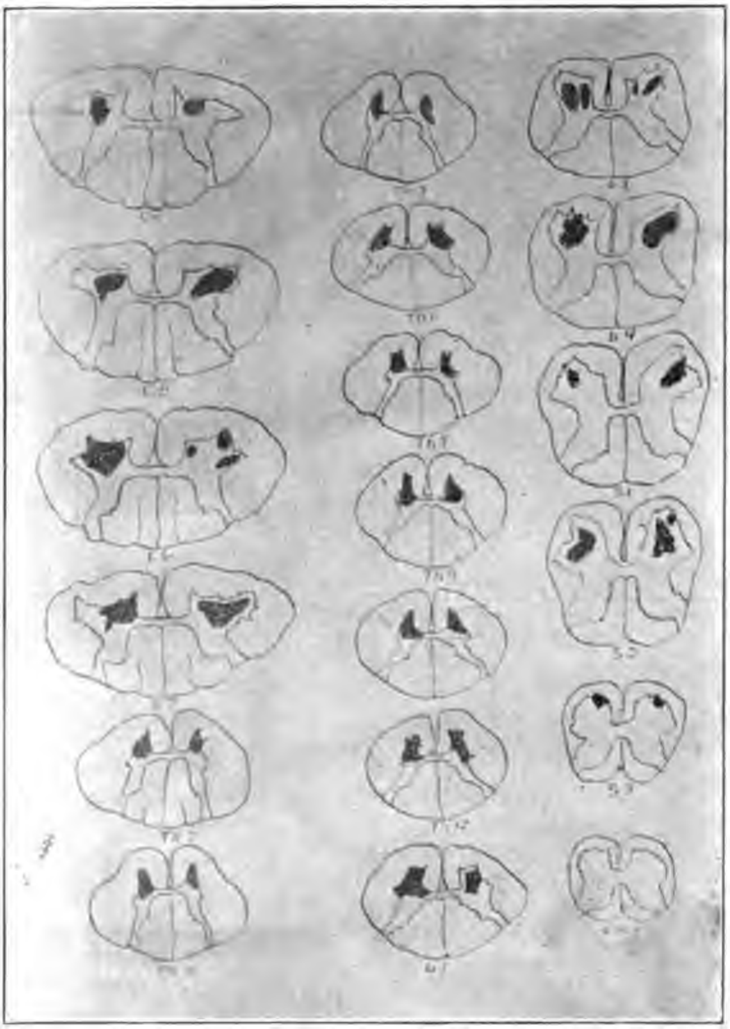

FIG. 1. Shaded portions represent the areas of destruction within the gray matter. Drawn by means of Edinger projection apparatus and photographed.

were extensively involved and, in order to prevent repetition, characteristic levels in the cervical, dorsal and lumbar segments only will be described, except where lesions show a marked departure from the common type.

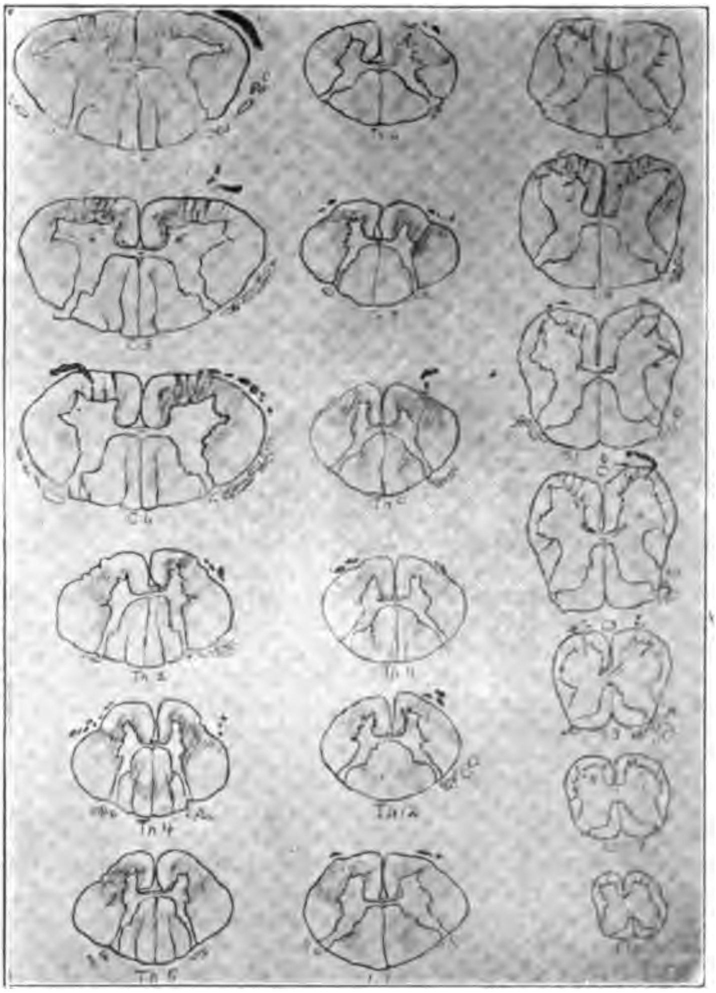

FIG 2. Shaded portions represent the areas of degeneration within the white matter. Drawn by means of Edinger projection apparatus and photographed.

The Cervical Cord.—The normal contour of the cord is lost.

The anterior horns are smaller than normal, causing a depression of the white matter over them. The disease process is bilateral, fairly symmetrical and limited in its active manifestations to the ventral gray matter. The anterior horns, however, are not wholly diseased but the lesions are distributed in irregular patches, which vary greatly at different levels. One group of cells may be involved in one segment and remain partially intact in another, while an entirely different area of the gray matter is affected.

Sections stained by Nissl's method show the following characteristics. With slight magnification the anterior horns can be identified only by the presence of very faintly staining cells within the gray matter, more especially near the periphery (hypertrophied neuroglia cells). A few remnants of nerve cells of the anterio-median group exist; in other levels a few cells remain in the antero-lateral group. The cells showing the least degree of involvement are in the intermedio-lateral group. In some sections no nerve cells can be found in the anterior horns and in general one is impressed with their great scarcity. Those that remain are coarsely granular and stain deeply. The chromatin appears heaped up in clumps and but little non-stainable substance is seen. The nuclei, when present, are round and located centrally and the nucleoli stain deeply. The cells are small, angular, flattened, long and spindle-shaped. Their prolongations appear suddenly broken off. No cells show any evidence of a chromatolysis. There is extensive hypertrophy and hyperplasia of neuroglia cells within the ventral gray matter. These cells are most numerous at the junction of the gray and white matter and are widely scattered throughout the body of the anterior horn. A few isolated hypertrophied neuroglia cells are seen in the periphery of the antero-lateral tracts in the white matter. A slight neuroglial proliferation is also evident near the base of the posterior horn. These hypertrophied neuroglia cells are large, stellate, and appear drawn out in places. The cytoplasm takes a faint bluish pink stain. The nucleus is large, pale and eccentric. Scattered among these cells are numerous normal glia nuclei. Specimens stained by the neuroglia method show a large increase in neuroglia fibers throughout the entire ventral gray matter, the anterior commissure and anterior root zone. The cytoplasm of the hypertrophied neuroglia cells does not stain by this method. The fibrils are short, coarse, straight and angular. They stain deeply, but seem to end abruptly, near the cell body, and at their periphery. Occasionally thin fibrils appear to mark out the boundary of the cells. Cells of this type are numerous in the immediate vicinity of the most pronounced vascular changes.

The blood vessels of the ventral gray matter show extensive changes. They are engorged with red blood cells and the walls are thickened. Many new vessels have been formed and in places the nervous elements are entirely replaced by a fibrillar network and distended capillaries. Branches of the anterior spinal artery and of the antero-lateral pial vessels are about equally affected. These vessels appear to lead directly into the patches of destruction within the gray matter, which may be considered partly as areas of anemic necrosis. These patches have a loose areolar structure and are made up of a fibrillar scar tissue with vascular proliferation. Nerve cells, nerve fibers and glia cells are absent, but newly-formed vessels are abundant. Occasionally these areas are crowded with polymorphonuclear leukocytes and small lymphocytes, but no foci are found which could be considered purulent.

The perivascular spaces of the pial and anterior spinal arteries leading toward these lesions are crowded with extravasated cells of various types, but only after they enter the gray matter.

Polymorphonuclear leukocytes are scattered throughout the areas of infiltration in small number. Small lymphocytes are closely packed about the larger vessels within the gray matter, four or five layers in depth. Within these areas a few isolated plasma cells are seen. The smaller capillaries within, and immediately surrounding the foci of destruction, show numerous characteristic plasma cells surrounding them. These cells have at times a mosaic-like arrangement and are usually oblong and angular with the characteristic appearance of the pale and eccentric nucleus and a heaping up of granules in the periphery. The larger and thicker vessels within the same areas are surrounded by an entirely different type of cell. A few plasm cells are present, but the prevailing type is a cell much larger than the plasma cell. It is round or oval in shape with a large, round, glassy nucleus, which shows a few deeply-staining granules The cytoplasm of the cell is very pale, and contains indistinct fine granules which are widely scattered. Occasionally slightly larger and irregular granules may be made out. By the Nissl method these granules appear of a purplish tint. Marchi's method shows the majority of them, at least, to be made up of fat or myelin detritus. Occasionally the cytoplasm has a vacuolated appearance and at times a cell can be seen with an inclusion of lymphocytes; in one instance two lymphocytes were included within a cell. These cells are likewise found scattered throughout the gray matter. They are probably of the compound granular type, called by some "granule" cells; by others "fat granule" cells. The vessels within the pia and anterior fissure are free from infiltration and evidence of thrombosis or embolism is nowhere present. The anterior spinal artery is never involved until it penetrates into the gray matter; but the pial vessels show a slight perivascular lymphocytic infiltration within the white matter, increasing in intensity as they near the gray matter. In some sections the anterior spinal branches appear normal and the patches of necrosis and scar tissue seem wholly dependent upon the disease of the peripheral vessels. Weigert's medullated fiber method shows the following condition:

There is distinct grayness of the anterior roots and a scarcity of fibers in the anterior root zones. The connective tissue is increased, especially near the gray matter. There are numerous large vacuoles within the antero-lateral white matter. The nerve fibers which remain are for the most part normal in size, but there are many smaller, and some very much larger ones. There is a marked scarcity of fibers in the anterior horns, especially in the antero-median part. The fibers which remain in the gray matter are thick, short and varicose. There is an extensive disappearance of fibers in the anterior commissure. The fibers entering the posterior horns have a normal appearance.

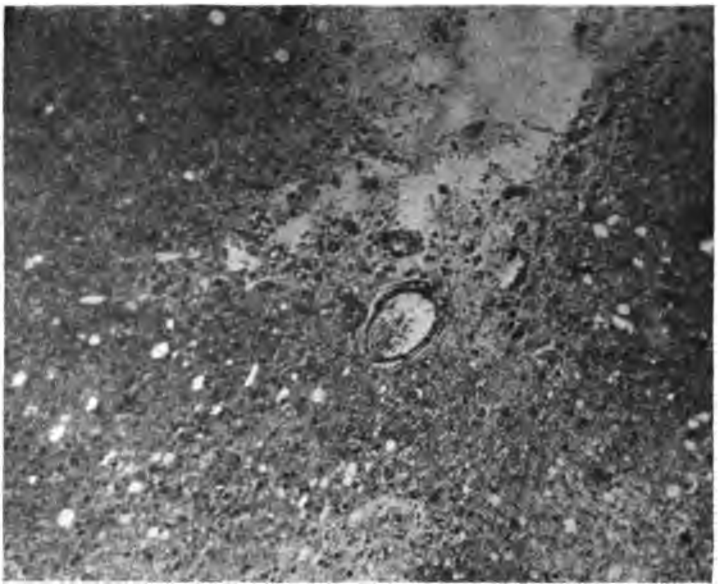

FIG. 3. Marchi specimen. Dilated peripheral vessel surrounded by cells with black granules. Disintegration of gray matter.

Marchi specimens show numerous black degenerated fibers in the anterior commissure, some within the gray matter, many throughout the anterior root zone, and all the white matter adjacent to the gray matter. Scattered degenerated fibers are seen in the lateral parts of the posterior columns. Most of the larger cells in the perivascular spaces, and scattered throughout the gray matter, show large numbers of fine and coarse fat or myelin granules.

The Thoracic Cord.—The anterior horns throughout the entire thoracic cord are extensively atrophied and completely devoid of nerve cells or nerve fibers. The diameter of the cord is greatly diminished. The gray matter of both anterior horns is entirely replaced by a highly vascular and loosely reticular scar tissue, the interstices of which are in some areas closely filled with polymorphonuclear leukocytes. A few fairly normal cells may be seen at times in the intermedio-lateral group. The vascular changes are as pronounced as in the cervical region and involve about equally the branches of the anterior spinal artery and the peripheral vessels. In some sections the alterations in the branches of the pial vessels predominate; in others, changes about the anterior spinal artery are most extensive. On the whole, the former appear to share in the inflammatory process to the greatest extent.

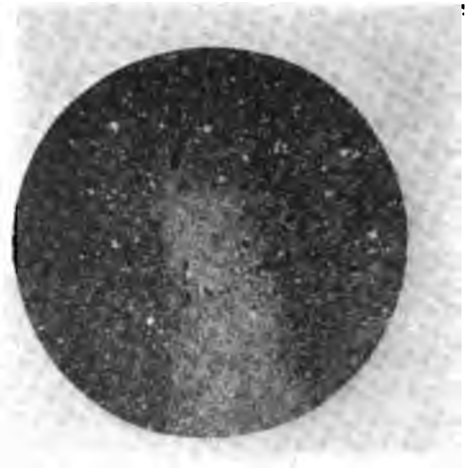

FIG. 4. Anterior horn of thoracic cord made up entirely of scar tissue and vessels.

A few hypertrophic neuroglia cells are seen, chiefly at the junction of the normal gray matter with the posterior horns. Nissl specimens demonstrate numerous cellular elements within the interstices of the scar tissue replacing the gray matter of the anterior horn. Many of these cells are plasma cells; some are of the compound granular type, while the majority are neuroglia cells and lymphocytes. A few isolated neuroglia fibers are seen within the destroyed areas. Where nerve fibers are present, they are short, coarse, stubby and varicose. The most absolute destruction of the ventral gray matter is at the level of the fifth thoracic segment.

The anterior roots are almost completely degenerated and the zone of white matter surrounding the anterior horns shows a great scarcity of nerve fibers. Only a few fibers can be seen in the anterior commissure. A similar, though rather less diffuse, area of degenerated nerve fibers is seen in the lateral parts of the posterior columns.

The cells of Clarke's column appear normal in number, but show distention, central chromatolysis, and a heaping up of granules in the periphery. The nuclei are eccentric. The network of fibers within Clarke's column is rather diminished in the upper part of the thoracic region.

The Lumbar Cord.—The destruction of the ventral gray matter is not as complete as in the thoracic region but has the same characteristic patchy arrangement that is seen in the cervical region. A few nerve cells in various stages of destruction are seen in the anterior horns; the remainder of the anterior horns is made up of numerous small deeply-staining neuroglia cells and many large, pale, hypertrophic neuroglia cells, especially in the vicinity of the larger vessels. These cells are also very numerous in the region of the anterior commissure. Few cells are seen in Clarke's column, and these have a normal appearance. The vessels entering the anterior horns from the periphery, especially with the anterior roots, show extensive alterations; some are very much thickened and surrounded by varying degrees of cellular infiltration. These cells are lymphocytes, closely crowded together. Aside from them there are a few scattered cells which answer the description of Unna's plasma cells, and some compound granular cells. Small clusters of nerve cells, which are small and irregular, are seen in the intermedio-lateral part of the anterior horn. The posterior horns are normal. Hematoxylin and eosin specimens show the anterior horns to be made up of a fine fibrillar network, and numerous large neuroglia cells with pale pink protoplasm.

Marchi specimens show degenerated areas corresponding to those in the cervical and thoracic region, but much milder in degree. There is an enormous new vascular formation within the anterior horns, especially in the antero-lateral part. The branches of the anterior spinal artery are somewhat distended, but the greater part of the vascular changes are derived from branches of the pial vessels. Minute hemorrhages and extensive perivascular infiltration are seen. The pia is slightly thickened. There is an extensive neuroglial proliferation in the anterior commissure and in the middle part of the gray matter. A few scattered neuroglia fibers are seen in the anterior horn where the destruction is most pronounced. Neuroglia cells are increased at the junction of the gray and white matter, in the white matter of the anterior root zone and in the periphery of the cord from the posterior horns forward.

Weigert's stain shows a great diminution of fibers in the gray matter surrounding the anterior horn. The anterior commissure shows more normal fibers than previous sections.

In the fourth lumbar segment many normal cells are seen in the antero-mesial part of the gray matter, and the foci of destruction are smaller and nearer the periphery. The vascular changes are confined almost wholly to the peripheral vessels.

Sections from the sacral segments show similar changes, though to a less degree. Again the vessels entering from the periphery, especially with the anterior roots, show the most extensive inflammatory changes. The foci of destruction are small and many normal nerve cells are present. The same hyperplasia and hypertrophy of neuroglia cells is present near the foci. The white matter shows no involvement.

A bacteriological examination was not undertaken, but no micro-organisms could be demonstrated in any of the sections.

Summary.—Clinically we are dealing with a strong and apparently healthy boy who, after a few days of general indisposition, suddenly developed a flaccid paralysis beginning in the legs and within 24 hours spreading to the arms. These phenomena were accompanied by a rise in temperature. After a brief, acute stage, gradual but slight improvement took place. After 13 weeks, death ensued from pneumonia. The onset was sudden. There was no history of preceding infectious disease or of exposure. As far as I am able to learn, no other similar cases occurred in Worcester at this time, neither were there any epidemics of cerebro-spinal meningitis or other infectious diseases. The presence of tubercular disease in a brother and sister is highly suggestive in view of the absence of any definite etiological factors, but microscopical examination failed to show any micro-organisms.

In summing up the anatomical changes we find throughout extensive alteration of the gray matter of the cord, more especially of the anterior horns. The posterior horns have suffered less although here and there slight vascular changes, as well as a mild neuroglial proliferation, are evident. The entire cord shows primarily a widespread inflammatory condition of the gray matter. The vessels are thickened, distended, engorged with red blood cells and increased in number. The perivascular spaces are crowded with cells, chiefly of the lymphoid variety. Plasma cells are fairly common and most abundant about the thickest vessels and around minute newly-formed vessels. In these regions there are but few lymphoid cells and the tissue destruction is most pronounced. The areas in which the lymphoid cells seem to predominate appear to be of more recent origin. They are also numerous in areas immediately surrounding the vessels as they enter the gray matter, becoming less intense as the destroyed areas are reached. Compound granular cells are found in some sections in fairly large numbers about the blood vessels and within the surrounding tissue. Large areas of extravasated leukocytes are found in the upper dorsal region, almost replacing the entire gray matter in some levels. Similar smaller foci are present in the cervical and lumbar regions, but no pus or abscess cavities are found. None of the vessels show any evidence of thrombosis or embolism. The vessels which participate in the changes are derived both from the anterior spinal artery and from the pia. The largest foci are found in the course of the branches of the anterior spinal in the cervical region, but the pial vessels show more involvement in the lower thoracic and lumbar cord. The perivascular infiltration is confined almost entirely to the gray matter though some of the vessels which enter from the periphery show a moderate degree of infiltration within the white matter, but increasing in intensity as they near the gray matter. In every section the anterior spinal artery appears normal until it penetrates into the gray matter.

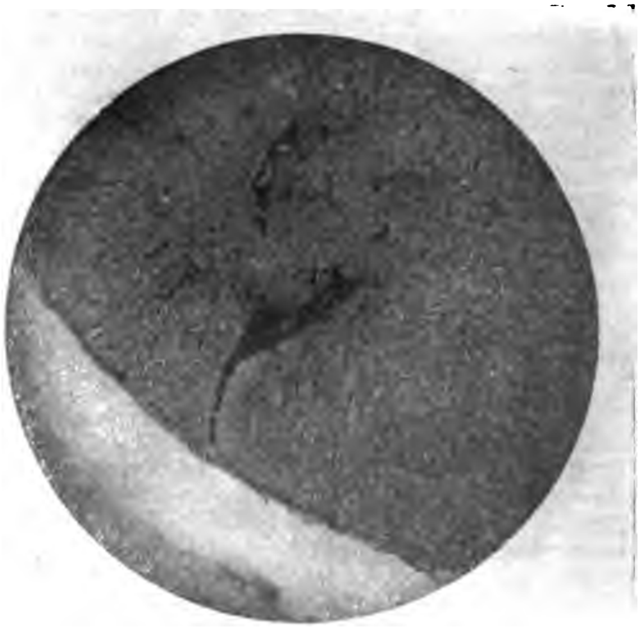

FIG. 5. Pial vessels showing extensive perivascular infiltration. Many newly-formed, thickened vessels in the gray matter.

The anterior horns are almost entirely devoid of nerve cells and fibers. Those cells which remain are seen chiefly in the extreme periphery of the gray matter, but in different areas at the different levels, apparently depending directly upon the blood supply. They have undergone great destructive changes; are smaller than normal; the processes appear suddenly broken off; the Nissl granules are arranged in large, coarse, deeply-staining clumps; the cells are angular, flat, narrow or spindle-shaped, as if they had been subjected to mechanical pressure. Very seldom a normal cell is seen while the majority are mere remnants. The group of cells which are most constant are the intermedio-lateral. In the thoracic region the anterior horns are made up almost entirely of scar tissue, very rich in vessels and without any nerve cells. Cells of Clarke's columns are not diminished in number but show the axonal reaction. The anterior horns throughout are atrophied, more especially in the dorsal region, causing a distinct flattening of the cord over them. The destruction is bilateral with practically complete involvement of the anterior horns of the dorsal region but with a peculiar destruction in small and large irregular patches in the cervical and lumbar regions.

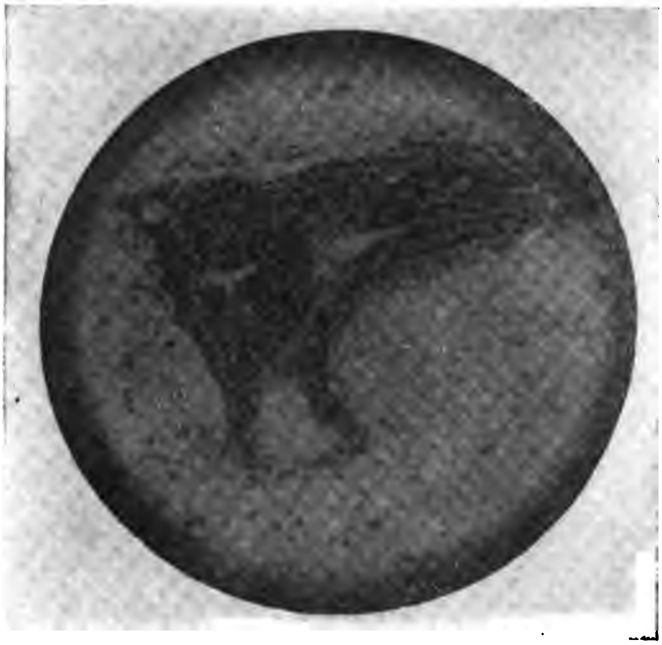

FIG. 6. Same vessel as that pictured, under higher power.

(*To be continued*)

EPILEPTIFORM ATTACKS DURING THE COURSE OF DEMENTIA PRECOX.

By Paul Masoin, M.D.,
OF BELGIUM,

PHYSICIAN AT THE COLONY OF GHEEL, BELGIUM.

Among 825 patients observed by Doctors Meens and P. Masoin there were 65 cases of dementia præcox. Of these 65 patients five only were subject at times to clonico-tonic seizures of varying character. The occurrence of such seizures during the course of this disease is rare, and their pathogenesis is still less understood. The following observations are only designed to furnish data for those who may later desire to take up the work of criticism and synthesis in further investigations along these same lines.

It need hardly be mentioned that these notes do not concern the occurrence of epilepsy which has not been previously diagnosed, ultimately resulting in dementia. In the cases reported the diagnosis of hebephreno-catatonic dementia (Meers) had been established beyond question, and the majority had been under observation for a number of years, two from the first onset of the disease.

Case I.—A., male, twenty-three years old. First onset of disease at the age of nineteen or twenty.

1901: Wandering and difficulty in holding the attention.

1902: Impulsive movements; progressive intellectual enfeeblement.

1903: Intellectual enfeeblement; stereotypie (tics); impulsive movements; tremor.

During the preceding months this patient has been subject to a kind of general convulsion. There was no fall nor loss of consciousness, but frothing at the mouth and convulsions of the face and limbs. The seizure was of short duration, much shorter than an ordinary epileptic attack. With a different order of symptoms this same patient has had two or three attacks resembling asthma with prolonged expiration, and the chest in forced inspiration. These seizures are probably of bulbar origin.

Case II[1]—B., female, aged thirty years. Onset of disease seven years ago following (?) an attack of typhoid fever. Negativism very marked. Frequent and violent impulsive movements.

In December, 1902, the patient suddenly gave a cry and fell to the floor. She appears to have had clonic spasms of the limbs, but without loss of consciousness. She arose without assistance, and there was no period of stertor, grinding of the teeth, nor bloody froth at the mouth. As soon as she was on her feet she repulsed the proffered assistance of those who wished her to sit down. It was impossible to find out the exact degree of severity of this attack, but it appears to have been of short duration, not more than two or three (?) minutes.

The attack just described was the only one during three years of the most minute observation, during which time at at least one special attendant constantly kept watch over the patient.

Case III.—C.,[2] female, twenty-nine years old. Onset about nine years previously, with stupor, very pronounced negativism, and muscular spasms. "This patient," says Dr. Meens,[2] "underwent nervous crises, generalized clonico-tonic cramps, during which sensation was retained, the attack lasting from one to several hours, in one instance for a space of two days. The face was distorted, lips pursed, arms thrown about and hands clenched, and the legs flexed upon the abdomen."

Case IV.—D., female, fifty-three years old. Onset of disease at the age of twenty-six. Hebephreno-catatonic form with frequent spasms. I was informed by those in charge of this patient that she had had a "convulsion [crampe] which seized her all over the body," but that it had soon subsided. These people, who were accustomed to handling epileptics, assured me that what they had witnessed was not an ordinary epileptic attack.

On June 11, 1902, the patient became restless, walked about incessantly, touching the furniture and the wall, moved the chairs, and frequently mounted the first steps of the staircase. Suddenly she became violent, uttered a scream and went into a convulsion, at this time giving all the evidences of a true epileptic seizure. A period of coma followed. When I saw the patient some hours later, she was calm, and there was no gnashing of the teeth, although there was continual tremor of the left foot.

July 14, 1903, the patient was in bed at 9.30 p.m., when those in charge of her heard a scream, and going to her found her in an epileptic attack, frothing at the mouth, with convulsions of both upper and lower limbs, eyes rolling and face contorted. This was followed by a period of stertor. I repeat that those in charge of this patient had for a long time been accustomed to dealing with epileptic cases, and constantly living among them, and they were certain that they had witnessed a genuine epileptic attack. About midnight they were again summoned by another scream, and witnessed a similar seizure, with all the same characteristics.

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[1] Clinical report in the Journal de Neurologie, Brussels, 1902.

[2] Reported in Bulletin de la Societé Médecine Mentale de Belgique, 1902, p. 135.

About 3 a.m. there was another attack, identical in character, and at 6.30 a.m., the patient was discovered in a state of profound coma, "without consciousness." A woman in an adjoining room said she had heard a scream about half an hour previously, so it is probable that the patient had been seized with still another convulsion. Later in the morning she was comparatively calm, and there was no gnashing of the teeth.

Case V.—Male, twenty-four years old. Onset of disease about three years ago. Negativism very marked, tremor of body. In 1902 epileptiform seizures began. Quoting from the notes kindly furnished by Dr. Meens: "The eyeballs were rotated obliquely upward, there were general spasms with loss of consciousness. Stupor followed the attack, usually lasting only two or three hours, but at times persisting for as great a length of time as two or three days. These attacks were repeated quite frequently, as often as every month."

Later Observations: The patient did not scream, but the eyes were fixed and staring, the face convulsed, froth at the mouth, tossing of the arms and a period of stupor after the attack. Sometimes several of these seizures followed each other in rapid succession, at other times there would be but a single seizure lasting perhaps an hour.

A certain analogy between the attacks suffered by the patients mentioned in Cases I, II and III can readily be perceived, and in the same way between those of Cases IV and V. Case IV is an excellent example of the transition between these two groups, since the manifestations of her attacks presented characteristics of both.

In Case I we have a general convulsion, of the most simple form, without a fall, and of very short duration. In Case II there is a clonico-tonic crisis, also of short duration, but with a fall. During two years of the closest observation, no similar attack was observed. In the history of the third patient (Case III) we have crises frequently occurring and lasting some time. In all three cases there was no loss of consciousness.

Case IV, as has been already noted, represents the transition stage between the preceding group and that which follows. This patient underwent one crisis, a "generalized convulsion," without loss of consciousness; later she had an attack presenting epilepto-convulsive features; still later she appears to have had a succession of several attacks, unmistakably epileptic in character, with loss of consciousness.

In Case V we have frequent attacks undoubtedly epileptic in their nature.

The presence of these peculiar features in the course of dementia præcox (called by Meens hebephreno-catatonic dementia) has been observed by Kraepelin, but we have been unable to find any studies bearing directly on this point, although they would be far from superfluous. The principal motive in making the foregoing observations public is the hope that others may be encouraged to bring forward similar data, and thus furnish material for critical analysis, especially from the point of view of pathogeny. An analysis of the pathological anatomy might also be of great value in this connection.

At the risk of appearing radical and premature I cannot refrain from comparing these peculiar manifestations with the other motor exteriorations of hebephreno-catatonic subjects, particularly the muscular paroxysms. The impulse to movement, the incessant activity shown by these subjects seems to arise from a real necessity, to be dictated by a nervous hypertension (called by Kahlbaum "Spannungs-irresein") for which these sudden and violent externalizations are the discharge.

With reference to the general considerations which I have developed concerning these motor symptoms of catatonia,[3] I would readily assign the origin of these symptoms to the basal and mesencephalic centers. All these clonico-convulsive symptoms, from the simple spasm to the characteristic epileptic attack, would be referred to a subcortical origin, in the corpus striatum, optic ganglia and medulla. The expiratory crises of A. (Case I) are a strong argument in favor of this view.

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[3] Journal de Neurologie, 1902, No. 4; Bull. de la Soc. de Méd. Ment. de Belgique, 1902, December; Congrès de Bruxelles, 1903.

PARAPLEGIA FROM FRACTURE OF THE FIRST, SECOND AND THIRD DORSAL VERTEBRÆ; SEVEN OTHER FRACTURES; LAMINECTOMY; RECOVERY WITH ABILITY TO WALK WITH ASSISTANCE.[1]

By Guy Hinsdale, M.D.,

OF HOT SPRINGS, VA.

MEMBER OF THE AMERICAN NEUROLOGICAL ASSOCIATION, AMERICAN CLIMATOLOGICAL ASSOCIATION, AMERICAN ACADEMY OF MEDICINE, AMERICAN MEDICAL ASSOCIATION; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, ETC.

I have pleasure in presenting the following history, not only because it is one of the most remarkable cases on record but because several members of this association have seen the patient with me at various times. The lessons which it has taught are not altogether of a scientific character, interesting as these are; there has been a remarkable exhibition of fortitude under the most desperate situations; patience, courage, hope, determination,—in short, the ideal attitude that shows strength of character when it is most needed.

Miss E. K. was injured July 25, 1901, at Manchester, Mass., by an explosion of acetylene gas. The accident occurred in the evening while the patient was lying on a sofa on the first floor of a country house directly over the gas machine. In the explosion the house was wrecked, the floor was thrown upward from its joists upon which the patient was found covered by the sofa, by a mantel, a Franklin stove and other debris. The house immediately took fire and was consumed, but the patient and other occupants were rescued all more or less injured or burned.

Miss K. was removed to an adjoining cottage where her injuries received immediate attention. They consisted of ten fractures including the first, second and third dorsal vertebræ, the sternum, right clavicle, right radius, left elbow joint, (badly comminuted with probably a dozen fragments); a compound fracture of the right ankle joint; fracture of the left fibula, a dislocation of the left radius at the wrist, a fracture of the nose, and a lacerated wound of the right brow.

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[1] Read at the meeting of the American Neurological Association, June 1, 2 and 3, 1905.

There were bruises in the upper portion of the back and sacral region. Dressings were made on the same night as the injury and also on the following day under ether.

There were no serious injuries to the head and the patient was conscious enough to call for help. As soon as she was carried to a place of safety it was noticed that she could not move her lower limbs. She was probably paralyzed immediately. She was attended by Dr. G. H. Washburn and Dr. Arthur T. Cabot of Boston. I saw her with Dr. Washburn, Dr. Cabot and Dr. G. L. Walton in consultation on July 28. She was then recovering from shock. There was complete paralysis for motion and sensation on both sides downward from a line drawn two inches below the nipples. Above this line was a zone of hyperesthesia. There was total loss of control and loss of sensation in the bladder and rectum.

The knee-jerks with plantar, abdominal and epigastric reflexes were absent. There was no irregularity of the spine, no prominence or any crepitus to indicate locally the seat of fracture, but there was extreme pain on passive motion and great sensitiveness over the third dorsal vertebra with soreness over the first and second dorsal spines.

We urged an immediate operation. This was performed on July 29 by Dr. Cabot assisted by Dr. S. J. Mixter and Dr. Hugh Cabot, and consisted of a laminectomy, removing the arches of the first, second and third and a part of the fourth dorsal vertebræ. There was a slight laceration of the dura. In removing the arches it was found that they were impacted as well as fractured, and the fragments were lifted out without much use of the bone forceps except for trimming. The opening in the dura was extended with scissors for one inch. Clear cerebrospinal fluid escaped. The cord was edematous, of a deep pink color, but to all appearances was not severed. A probe was passed up and down below the dura to detect any further cause of pressure. Nothing further being found, on the advice of Dr. Walton and myself the dura was not sutured. The wound was then closed with provision for drainage. This was removed on the following day under ether and the dressing replaced on alternate days; the cerebrospinal fluid escaped freely.

On the second day after the operation there were convulsive movements of the hands and forearms. They were very slight at first, infrequent and painless, and not attended by any loss of consciousness. As the attacks were repeated they rapidly increased in violence and extent, involving the forearms, shoulder girdle and later the neck, face and entire head, but always limited to the parts which had escaped paralysis. There was no loss of consciousness at any time. The movements came on suddenly during sleep or while the patient was awake. They agitated and unnerved her. They were sufficient to wake her violently and were usually controlled by firmly grasping the hand, arm or shoulder or steadying the head with both hands. The slightest motion of the head or neck would sometimes bring on the movements.

By the sixth day after the operation breathing was difficult and there were neuralgic pains about the heart with spasm of the diaphragm. These attacks demanded the free use of morphine hypodermatically. Nine days after the operation there were bilateral grimaces with up and down and lateral movements of the jaw. They were very rapid, and were attended by rotation of the head and movements of the laryngeal muscles. The sounds were rather alarming, and the breathing was rapid as in hysterical breathing. There was great agitation. The patient was uneasy about the face. There was no actual difficulty in swallowing, but the mouth was dry. There was never any temperature above 101.5° F., and as the flow of cerebrospinal fluid ceased the movements subsided. Codeine, atropine, sodium bromide, and camphor monobromide were also used during the first two weeks.

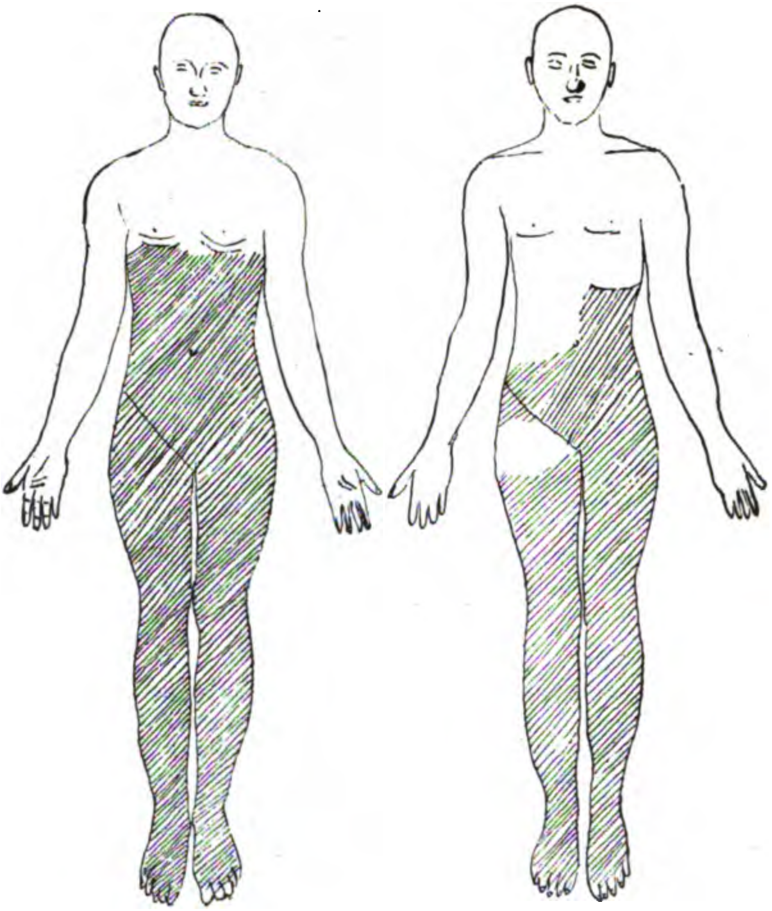
During this period the following notes were made as to sensory and motor symptoms: There is a dissociation of sensibility:—total paralysis to pain below a line drawn two inches below the nipples; above this a hyperesthetic zone of about an inch and a half or two inches; thermoanesthesia over the area of analgesia. Tactile sense preserved over most of the abdomen, thighs, feet and toes.

Motor symptoms: Power lost for all muscles supplied below the first dorsal segment. Bladder and rectum paralyzed. Catheterization. Bowels moved on alternate days by enema. At times involuntary evacuations. Pulse 88-100. Digestion good; nutrition and general color well maintained.

Profuse cold perspiration about the head and neck a marked symptom. Urine 40 to 48 ounces daily. The normal character of the urine was maintained.

Knee-jerks absent for the first month; returned August 28. Babinski reflex noted at the end of one month on the left side first. At the end of seven weeks there was no ankle clonus in either foot. There were increased knee-jerks in both sides and marked and continuous patellar clonus of both legs.

During all this it was a most difficult matter to handle the patient, to dress the various fractures, to feed her and to secure free bowel movements, and to guard her from infection of the bladder. A Crosby bed permitted a proper dressing of the spine, attention to the back, bowel movements, etc., without any change in the general position of the body. This bed was indispensable as it allowed the patient to be supported on horizontal bands when the mattress and sheets were removed from below. Notwithstanding the greatest care and the use of air cushions it was not long after the accident before a slough formed over the sacrum chiefly on the right side. An indurated area on the left buttock broke down and communicated with the opening of the first slough. This was laid open October 20 and granulated; the original cavity healed.



The shaded lines in the figure on the left represent the anesthesia to pain and temperature a few days after the injury. The shaded lines in the figure on the right represent the anesthesia to pain and temperature three years and four months later.

The superficial temperatures were taken by Dr. Walton nine weeks after the accident as follows: Dorsum of right foot

94.6 F., left 95; front of right thigh 92.5, left 92.5; dorsum of hand R 90.8, left 91.2, upper arm right 91, left 91.2; right side of neck 94; right cheek 93.2, left 92.5; temperature by the mouth, 98.4 F. This shows vasomotor paresis and the accompanying dryness of the skin shows lack of sweat nerve innervation.

At the end of eight weeks ankle clonus and Babinski reflex were well established; at the end of nine weeks the patient could flex the left thigh voluntarily, and two days later she could separate the knees and also extend and flex the left toes fairly well; she was slightly able to flex the right toes.

The patient was removed to Philadelphia on October 3. On November 12 it was noted that the general morale, nutrition, color and sleep were excellent. The area of analgesia and thermo-anesthesia was lower by six or eigth inches anteriorly and posteriorly. The patient was able to use her spinal muscles better so that she could arch her back a little when the draw sheet was adjusted. Reflex movements of the lower limbs were noted. These were excited by any irritation or might be spontaneous. A spastic condition developed with strong adduction of the knees with "lead pipe rigidity," rendering catheterization difficult, and when the knees were flexed requiring considerable force to straighten them. Ankle clonus was easily elicited; Babinski reflex was marked. Plantar reflex was present. Voluntary motion was possible, in the left limb but not in the right, except in the great toe.

Treatment: Massage and faradism on alternate days; 10 grains of potassium iodide and 1-24th grain of protiodide of mercury t.d.; 5 grains of amidoform for the prevention of bladder infection; 1-24th grain of bichloride of mercury was employed for a short time.

Seven and a half months after the accident the spastic condition of the lower extremities did not seem to have abated and after consultation with Dr. Weir Mitchell, Dr. DeForest Willard and Dr. H. R. Wharton it was decided to cut the tendo Achillis on the right side. This was done under ether, and the foot was placed in a plaster bandage and held in flexion. The sphincter ani was stretched on account of spasm of that muscle. The operation was followed by heightened reflex excitability, which chloretone and sodium bromide in large doses controlled fairly well. There was no unpleasant effect from the ether. The tension of the calf was entirely relaxed, but the limb was repeatedly raised in bed involuntarily even during sleep. This operation was followed a fortnight later by section of the other tendo Achillis and the adductors of the thigh at the pelvis. The lower extremities were placed in plaster bandages and held wide apart by sand bags and bandages. While under ether the clonus was marked in the left foot and there was a good deal of twitching of the limbs after the operation. On December 27, 1903, the hamstrings were cut on both sides. This was the seventh time the patient was under ether since the accident.

One year and nine months after the accident the dissociation symptom was still present. Analgesia and thermoanesthesia below a horizontal line three inches below the breasts and posteriorly from a point twelve inches below the last cervical vertebra. The buttocks, particularly the right, had lost entirely the sense of heat and cold. On the outer side of the leg there was confusion of sensation, heat being interpreted as cold; there was also some slight sense of pain on the outer surface of the right thigh.

In the left heel temperature sense was reversed; in the dorsum of the foot there was no distinction between heat and cold. In the dorsum of the right foot temperature sense was lost, but in the sole cold and heat were recognized slightly, cold better than heat.

Motor symptoms. Patient could flex the left thigh, leg and foot at will. In flexing the left knee the right limb frequently followed involuntarily. She had a sense of pressure as of a bandage over the left knee. With the aid of braces for both limbs arranged to support the thigh and leg, and with a broad leather corset with supports under axillæ, both being attached to rollers moving in a trolley fixed to the ceiling of the room, she had been able to practice walking. She was able to rest nearly her whole weight on the left foot. By the greatest persistence, not by daily practice but, on account of the great exertion, at intervals of one, twice or three times a week she practiced the voluntary exercise of her limbs. At this period she could flex and extend the right thigh, leg and foot but more feebly than the left. The extensor of the right foot was not very satisfactory. She could flex and extend the toes better on the left than on the right. There was a lead pipe rigidity in the right limb, but not in the left. Adduction and abduction were better in the left than in the right.

Reflexes: Knee-jerk increased on both sides. Ankle clonus in the right, moderate; not obtained in the left. Clonus of the great toe on both sides. Babinski, present. Sleep was frequently disturbed by spasmodic movements of the limbs.

Two years and three months after the operation there was still a good deal of spastic rigidity in the right leg. Knee-jerk plus; Babinski present; ankle-clonus hard to elicit, confined chiefly to the toes. The needle point produces involuntary movements. Patient feels the needle (tactile sense) but not as pain. In the left leg the knee-jerk was greater than in the right. There was a slight clonus which ceased quickly. There was good power of flexion and extension of the hip, knee and ankle joints. Babinski present. All the tissues were firm. In general there was less tendency to excessive perspiration. The cicatrices in the upper dorsal and sacral regions were not tender on pressure. There was a region below the right scapula and toward the axillary line very sensitive to touch and pressure and painful at times without pressure.

The patient had been able to use a wheel crutch and could stand alone in the crutch supporting the weight of the body with the arms extended over the head. She had gained strength and weight.

Condition three years after the accident: General condition and color good. Weight 143 lbs. She can turn over in bed from either side without help, and is gaining in strength. She walks in the wheel crutch and can get from the bed to the crutch with a little help. Her arms are very strong, and there is free motion in the left elbow though extension is not full.

Sensory symptoms: Touch sense normal. Pain sense acute over the right side of the trunk, upper thigh and outer side fading toward the middle; present also on the inner side of the thigh high up. Left side: Touch sense normal; moderate pain sense over the thigh; also on the leg; not on the dorsum of the foot, but present in the sole.

Reflexes: Knee-jerk plus; ankle clonus present, Babinski present.

The bladder resumed its normal function three years after the accident. Not once in all that time was there any infection, the nurse using a glass catheter, the same instrument and the same nurse having been in attendance for over three years.

Regarding the various fractures of the extremities the results are far better than could have been anticipated. She uses all her joints freely excepting some restriction in extension and flexion of the left elbow joint. The very slight deformity does not inconvenience her, the right ankle joint where the compound fracture existed shows a good result.

One feature of the case was the excellent maintenance of muscular tone by massage and the general restoration to usefulness beyond what seemed possible. The patient can take long drives and does not show fatigue or any tendency to weakening of the spinal column.

The dissociation symptom may also be mentioned. This is not an infrequent symptom in injuries of the cord at this level. The dissociation symptom always suggests syringomyelia. Ten years ago Kocher[2] and later Lloyd, Rhein and Gordon have reported cases of traumatic affections of the spinal cord simulating syringomyelia.

The occurrence of convulsions after laminectomy where the spinal fluid is allowed to escape was noted in a case operated upon by Dr. Edward Martin at the Hospital of the University of Pennsylvania three years ago. This was a patient of Drs. Spiller and Musser. In this case after emptying a cyst of the spinal cord there was a gush of cerebrospinal fluid followed by clonic spasms. Because of this the dura was completely closed by suture and the external wound was sewed up without drainage. In this case the spinous processes and laminæ of the first, second and third lumber vertebræ were removed.

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[2] Kocher. "Verletzungen der Wirbelsaule," p. 553. *et seq.*

In the case which we have reported the spastic spinal paralysis was a residuum of a myelitis or a pressure myelitis due to violence; there was total paralysis at first, but not a permanent break in conduction, the dissociation symptom being a conspicuous feature. The vasomotor disturbances of the head were due to the lesion of the first three pairs of dorsal nerves, for the dorsal cord is the principal seat of the vasomotors.

SMALL ROUND CELL SARCOMA OF THE SPINAL COLUMN.

By M. A. Bliss, M.D.,
of St. Louis.

Male, age twenty-two, dentist, single, height above five feet, eight, weight 145. Father, mother, one brother and two sisters living and well. The subject was the youngest of the family. Diseases of childhood were passed without disaster. Typhoid five years ago, no sequelæ. Only moderately robust but in good health up to beginning of last illness.

April 25, 1904, he applied to Dr. Homer Davis of Alton for relief of pain on movement in the cervical and lumbar spine, which he attributed to a cold. On the 28th the back was better but the neck was stiff and sore. May 10th there was much pain in the neck muscles. He used his hands to carry his head when changing position. The back gave no trouble. There was tenderness on deep pressure over the left posterior neck muscles, but none over cervical spines. A little later there was protrusion of the lower jaw so that the inferior incisors articulated in front of the superior. This could be corrected with a little effort and pain disappeared for a period in June and later returned.

May 20th his back became lame again and caused him much pain on voluntary motion or when he was moved, but he was always quite comfortable when lying still.

During the time above described there was no deviation of pulse or temperature from the normal, appetite was excellent. Patient examination failed to reveal anything abnormal in any organ of the body. At times improvement would occur in the neck so that he could move it freely and at times he could move his back muscles without pain. He was always free from pain when at rest.

June 23d was taken out of bed and placed in a Morris chair, but was so uncomfortable that he was almost immediately returned to bed. He screamed with pain on being moved, and following this excursion he complained of pain and coldness of the knees, lost all motor power and soon all reflexes. All sensation was much lessened up to the knees.

Retention of urine occurred, the bowels moved only with enemas. The anesthesia progressed upward but was rather higher on left than right side and became more nearly total.

There was no deformity of the spine to be made out at this time (July 5th) and an attempt at lumbar puncture was rewarded with only two or three drops of clear fluid of doubtful origin. There were no cranial nor cervical nerve involvements at this time. The grasp was good and even on the two sides. The mind was entirely clear. No pain was felt when he was at rest. From the 6th to the 11th of July, a swelling which had been noted in the right side of the neck for some days previous to first named date, increased rapidly, fully doubling in size, and the forward protrusion of the jaw recurred. The anesthesia had crept up, eight inches above the knee on left leg and four inches above the knee on right. On the 12th of July Dr. Mudd made an incision into the swelling on the neck and microscopical examination of a piece removed showed small, round-celled sarcoma. At this time a growth pushing downward and forward in the naso-pharynx, was made out, and the patient breathed though the mouth. He was able to take some solid food.

A few days later the left pupil contracted, and this and a slight ptosis of left lid were the first signs of involvement of any cervical or cranial nerve. The tongue then became disabled and speech was very indistinct. Anesthesia developed over the left side of neck down to the clavicle. Breathing became labored. On August 1st patient was bright, and at 3 A.M. brushed his own teeth. At 5 A.M. he lapsed into unconsciousness and died two hours later.

Post mortem examination 13 hours after death, was limited by request to the spinal column. On turning the body over on its face a hump was noted in the lower dorsal region, and an enlargement or swelling on the left side of the neck at the cranial base. An incision was made from the external occipital protuberance to the sacrum. With some difficulty the laminæ in the dorsal region were cut and the canal laid open.

At this point the bodies of several vertebræ were found disintegrated, causing the sound vertebræ to come closer together, overlap, and produce an angularity in the cord.

The cord and its membranes were removed intact and the intervertebral cartilage between the eleventh and twelfth was removed without destroying its dural attachment. This cartilage seemed to be in the midst of the growth and tumor tissue was found on the dura in this region. The neck swelling consisted of about half a pint of necrosed and tumor tissue. The anterior portions of the cervical vertebræ were disintegrated in the same way as the bodies of the dorsal and in much the same condition. Portions of the growth were visible on the dura, but at no point in either the dorsal or the cervical region did the cord seem to be involved.

No sections were made of the cord for the reason that it made, with its membranes, and the attached intervertebral disc, a beautiful and instructive macroscopic specimen, and because it appeared without question that the paralysis was due to the change in the relationship of the vertebræ to each other, caused by the disintegration of the bodies.

When the patient was first seen by the author he was sitting propped up in bed, with the head protruded forward, and presented the appearance of a pachymeningitis hypertrophica cervicalis. The swelling on the neck at that time was only such as to suggest an inflammatory process.

No deformity was noted in the lumbar spine and the explanation of the paralysis and anesthesia was not easy. Later when the sarcomatous process had produced further destruction it was apparent that the two involvements, cervical and dorsal, were of the same nature. But it was not until postmortem examination was made that the mechanism of the cord damage was understood.

It is possible that had an X-ray picture been taken on the occasion of our first examination (July 5th) that a gap might have been noted in the bodies of the dorsal spine, and I would suggest this procedure in a future case.

CASE OF FAMILY ATROPHY OF THE PERONEAL TYPE.

By G. L. Walton, M.D.,

of Boston.

This case accentuates the difficulty in classifying the so-called peroneal type of progressive muscular atrophy.

The history of the family and the physical examination (aside from the reflexes) of the patient who presented himself at the Neurological Department of the Massachusetts General Hospital, would strongly suggest that in his case at least, we have to do with a dystrophy rather than a spinal (or neural) type of atrophy. At the same time the diminution of deep reflexes in muscles apparently normal, suggests the possibility of a wider distribution of the underlying pathological condition.

The boy is 15 years of age. He has a brother of 25 who commenced to show a difficulty in walking and wasting of leg muscles at 11. The disturbance progressed in his case very slowly, finally attacking the upper extremities so that the muscles of the hand are now wasted and the movements awkward, but this older brother is still able to work as gate tender, 14 years after the onset of his disease, and with no suggestion of bulbar involvement.

Another brother of 18 has similar trouble in the legs of some years duration, and of gradual onset.

A third brother of 12 has complained for some months of slowly increasing difficulty in walking, resembling that of our patient.

A great-aunt on the maternal side is said to have become invalided by a similar trouble. No other instances are known in the family. Three sisters and one brother are unaffected.

In our patient impairment in the use of the legs appeared at about 11, and has been slowly progressive. There has been no pain, no paresthesia, no numbness, no further complaint of any kind.

Physical examination shows a boy rather delicately built but of fair muscular development. The face is somewhat pear shaped and symmetrical, the cranium is somewhat tigonal, the ears are fairly well shaped but rather prominent, the left more so than the right. No other signs of deviation are noted. There is no lordosis. The pupils and heart and arteries are normal. There is no involvement of cranial nerves, or of the muscles of the trunk, shoulder or upper extremities.

The gait shows a moderate tendency to toe-drop, especially on the left. Both feet are in the position of cavus. All muscles of the feet and toes are impaired, particularly in the peroneal distribution. All movements of the thighs are strong, and the nutrition of the thigh muscles is good without suggestion of muscular, adipose, or fibrous enlargement. The calves are comparatively small, the left one-quarter inch smaller than the right. The legs each measure 32½ inches from the crest of the ilium to the inner malleolus. The skin is everywhere normal, the feet are rather cool, the right somewhat cyanotic. *No trace of fibrillary twitching appears on prolonged examination.*

The epigastric, abdominal, and cremaster reflexes are lively, the plantar reflex consists of flexion, very slight in degree. The Achilles reflex is absent on both sides; the knee-jerk is extremely sluggish. Sensation is everywhere normal, including the thermal and pain senses. The muscles of the lower leg show very greatly diminished reaction to both the faradic and galvanic currents without qualitative change.

CHICAGO NEUROLOGICAL SOCIETY.

February 16, 1905.

The President, DR. HAROLD N. MOYER, in the Chair.

A Case of Idiopathic Muscular Atrophy with Bilateral Ptosis and Complete Ophthalmoplegia Externa.—Dr. Geo. W. Hall presented this case, stating that the patient, a boy, was 11 years old. According to his mother, he had been perfectly healthy until four years ago, when he had scarlet fever followed by gradual increasing difficulty in walking. The condition of the eyes had never been noticed by her. The family history was negative. The case approached the Landouzy-Dejerine type of the disease, but the boy can close his eyes and mouth very well. The reaction of the eyes to light and accommodation was normal. There were no fibrillary twitchings, and the muscles responded to faradism. Patellar and radial reflexes were present, but decreased. There was an absence of sensory and sphincter disturbances. Typical method of rising from the floor was demonstrated. The chief point of interest in the case was the presence of the bilateral ophthalmoplegia externa, which was probably congenital, due to nuclear involvement.

Dr. L. F. Barker said the case was interesting to him as to the exact relation of the ophthalmoplegia to the muscular atrophy. If it is primary in the eye muscles it is the only case on record. Ophthalmoplegia is not a part of general myopathy. The fact that it is so complete in this case seemed to make it very probable that it is binuclear in origin. One or two cases of myopathy have been examined by taking out a bit of the muscle and are said to have shown a typical change, though but one muscle was paralyzed. Here we have complete bilateral paralysis. It would hardly be supposed that some of the face muscles would not be involved. Dr. Barker leaned to the view expressed that this case was a combination of nuclear disease with the myopathy. The ocular signs may be congenital, or may have followed the scarlet fever. He did not think this could possibly be anterior horn disease, from the general distribution of the atrophy.

Dr. Harold N. Moyer said it seemed to him the distinctions made in these clinical types were somewhat involved. In some of these cases some muscles show hypertrophy, while others show the opposite, and it never had seemed to him a well-founded distinction. In this case, as in most others, can be seen two types mixed. Dr. Hall added that the boy had had unresolved pneumonia for some time, and there was still evidence of the old inflammation on the right side. In these cases it is not uncommon to find chest inflammation, owing to poor muscular action.

A Case of Intermittent Claudication.—This was presented by Dr. James B. Herrick. The patient had first been seen by Dr. Jacques, to whom credit should be given for the diagnosis. The patient is a Hebrew, 38 years old, a peddler, with no history of excessive alcoholism, no history of syphilis or over-indulgence in tobacco. He first noticed something wrong with the foot about a year ago, when he had some pain in the sole of the foot in walking, and the calf of the leg would pain and become stiff and cramped and the right foot would become almost as though dead, and white. Four months later a somewhat similar feeling was experienced in the left foot. There was the same painful sensation on elevating the foot, and the same loss of color. If he allows the foot to hang down it becomes more or less red, much as in erythromelalgia. Some time ago, what was supposed to be little abrasions appeared on the outer and inner surface of the right foot. These increased in size and there was more or less burning in and around these ulcers. Later the nail of the little toe became affected and loose, and a small ulcer appeared on the inner aspect of the little toe. A few weeks ago, in the night he was taken with a good sharp pain in the pericardiac region, running out into the arm, which resembled in some respects angina pectoris.

The physical examination, with the exception of that of the feet, is practically negative, but the radial pulse is small and of low tension. The registration is, in the right pulse, 120 m.m. of mercury, and in the left 90, instead of the average 140. No arteries can be felt in the foot. The dorsal posterior tibial arteries cannot be palpated, and nothing approaching sclerosis can be made out in the radials. Even in bed and when quiet the patient had more or less pain in the foot. The same phenomena of paleness and pain in the foot can be produced by pressure on the femoral as he lies in bed. In walking, one of the chief complaints is the pain in the calf of the leg. The case seems to be one of intermittent claudication.

The cases that have been described have most of them shown arteriosclerosis, both in amputated limbs and in autopsy. In this case no arteriosclerosis can be observed, but in some of the cases of Charcot and Erb there was complete absence of pulse in the foot, and this patient shows, as do many cases, either gangrene of the toe or some trophic disturbance. The relation between this and erythromelalgia would seem to be close. Erb, in the conclusion of his longest article about five or six years ago, called attention to the fact that he would regard intermittent claudication as angio-sclerosis, and that Raynaud thinks it is angio-sclerosis, that erythromelalgia was a neurosis, and that instead of a spastic condition there was rather the opposite, so that when the foot was down the dilators of the vessels, or the constrictors, were paralyzed and increase in color, and even in the temperature of the parts could be made out. Oppenheim, it was believed by the essayist, called attention to the fact that there was probably a marked spastic condition in these cases, and even Erb, who is inclined to call the disease a dysbasia angio-sclerosis, thought the same. One must assume a spastic condition to account for the bloodless condition and for the pain and gangrene sometimes met. An X-ray picture was taken of this foot in order to discover some sclerotic condition of the vessels, but it was entirely negative.

Asked by Dr. Billings as to the effect of the dilators, Dr. Herrick said that nitro-glycerine gave the patient relief from pain. Perhaps he had not been justified in not having given strophanthin, as he had wished the case to be seen as it was. Digitalis would be indicated, but it has a tendency to contract the vessels. If the heart beat could be materially strengthened, it would seem that some material good might be derived. Erb, in his case, had much benefit from strophanthin, as well as from the use of warm foot baths, electricity and iodides.

Dr. Mettler asked the effect of extreme cold and heat. The patient said he was worse in cold weather. Dr. Mettler said he had seen the statement in some place that the difference in one of the symptoms in erythromelalgia and Raynaud's disease was the difference in the sensations produced by extreme cold and heat. In the former cold gave relief, while the reverse was the fact in Raynaud's disease.

Dr. Herrick said he mentioned the race because Oppenheim is inclined to think the neurotic element plays quite a part, and the Hebrew race is notoriously neurotic, and he thinks that is why such a large per cent. of those who have this disease are Hebrews. Erb lays great stress upon tobacco. Another point, the patient has a right inguinal hernia, and in one case the wearing of a truss was regarded as having something to do with the obstruction of the circulation. The rectum is entirely negative, which ought not to be overlooked, as there might be a possibility of pelvic conditions causing obstruction to the vessels.

Dr. Chas. L. Mix said the case reminded him of one of his own, a man

;2 years of age, who had a chronic interstitial nephritis and a certain amount of arteriosclerosis. It began as this, with pain in the great toe, followed by whiteness of the toe and ulcers at the side of the nail. He did not lose the nail, but came about as near it as this patient. In his case the foot was better when elevated. The pain was worse at night than in the daytime. The patient liked warmth and the room was kept hot, and clothes heated on an oil stove were applied constantly to give relief. The pain was intense. At one time the necessity of amputation of possibly the whole leg, on account of the gangrene which seemed to be appearing, was considered. He was given nitroglycerine, spartein and atropine. He was on iodide of soda, also, continuously, and in the course of five months made a recovery. He has remained well, and had no return of his intermittent claudication.

Dr. Williams asked if there was evidence as to whether dilatation of the heart was myasthenic. The attack of pain which was suggested as having been angina might have been due to either factor and the feebleness of the pulse everywhere is not like the condition met in cardiac myasthenia; and yet there is no condition in the vessels, as far as can be seen, to make one think of cardiac degeneration. The pathology in this puzzling case may be elucidated to a single degree by the treatment. Attention was called to a paper by Thompson, of New York, on the effect of different drugs on relaxation of arteries, in which most remarkable results were secured by small doses of aconite where the nitrites failed.

Dr. Herrick said his plan was to give strophanthine to improve the cardiac tone. Erb calls attention to the local trauma occasionally present in these cases. One was the case of a well-to-do man who, about sixty days in the year, went fishing in the mountains and waded for hours in the cold mountain streams fishing for trout. He thought that determined the excessive degree of sclerosis in the foot. Dr. Bevan had called the attention of the essayist to the fact that Dr. Gunn, in his lectures, when he would meet with a case of Pott's toe would refer to the fact that "this man complains of tire in his legs, which is the tire of approaching gangrene."

Dr. Mix thought the fact that the pain is worse at night had some bearing on the etiology, as pointing to an increase in pain at the time of a decrease in the amount of arterial pressure, and the same had occurred in his case, *i. e.*, the maximum of pain at the minimum arterial pressure.

A Case of Ophthalmoplegia with Implication of the Motor Branch of the Fifth Nerve.—This was also shown by Dr. Herrick. The patient, a woman 32 years old, came to the Presbyterian Hospital three weeks previously. There is a negative family history, and her personal history rather uneventful, with the exception of the removal of a mass of supposed tubercular glands from the left side of the neck ten years ago. She also had several attacks of tonsilitis. The 10th of November of last year she worked in a cold room bending over a packing trunk, and took cold. She felt well on retiring, but on awakening noticed that she could not open her left eye, had quite a severe pain in the left side of the face, the jaws were stiff, and she could hardly open her mouth. The severe pain in the left side of her face lasted some time, as did the stiffness of the jaws, but with the relaxation of the jaws the pain disappeared, although she did not regain the ability to open the eye. She had no difficulty in swallowing, in talking, no trouble in seeing out of the right eye; taste, so far as she could tell, was perfectly normal.

She has a marked ptosis on the left side. She can wrinkle the forehead, and the movement of the facial muscles is perfect. The left eye is in a completely immobilized position, with the slight exception that when asked to look down there seems to be slight motion, with a slight rotation, a feeble motion of the superior oblique; this has occurred since her stay in the hospital. On the first examination there seemed to be absolute loss of all motion in all the eye muscles. There is complete absence of the reflex or contraction of the iris for accommodation and for light. There seems to be a very slight consensual reflex. The ciliary accommodation is entirely gone. With the right eye she can read fairly well, sees at a distance, but as things come nearer they appear less distinct. Examination of the retina shows slight pallor of the right disc, and more marked pallor of the left, and Drs. Hotz, Brown-Pusey and E. V. L. Brown agree that there is some pallor of the disc. The color field was narrow for green. The paralysis of the third, fourth and sixth seems to be complete, with the exception of a slight power of control over the superior oblique. It will be remembered that at the beginning of the attack there was quite severe pain in the left side of her face, seeming to be some irritation of the sensory fifth; also, difficulty in opening the jaw and irritation of the motor fifth. Now there is loss of power of the motor fifth. Sensation is perfect. When asked to close the jaw the masseter on the right side bellies up fully, but not on the left side. The same is true of the temporals. On opening the mouth wide there is little deviation to the left, the whole jaw being drawn a little more to that side. There is a little sagging of the floor of the mouth. There is a trace of albumin in the urine. Since the onset of this trouble she has had one or two attacks of tonsilitis, but none preceding this attack. There was no fever at the time of her taking cold. However, the next day after the onset she vomited, also for a couple of days thereafter.

The lesion would appear to be in the lower neurone, either as a multiple neuritis or some pressure at the base of the brain, as from nuclear disease of the pons, the latter being most probable. If it is agreed that she has optic atrophy the question can properly arise as to whether she has the beginning of some disease like multiple sclerosis, and that this is but an incident of multiple sclerosis. The knee-jerks are present and her station is good.

In the discussion of this case Dr. L. F. Barker said that, while interested in the case, which he had been permitted to examine,, he was not ready to make a full and positive diagnosis, but he had ideas which he was willing to express merely tentatively. Five or six possibilities may be thought of: first, some infection or intoxication may have caused a nuclear disease or a neuritis; the acute onset, the albuminuria, the recurring tonsilitis, and the fact that the patient has a little fever every day, though the temperature has never been higher than 99.8, suggest this. The leucocytosis amounted to only 11,000. Cases of ptosis and ophthalmoplegia something like this have occurred after common colds, rheumatism, influenza, diphtheria and tubercular meningitis. As the essayist has said, multiple sclerosis has also to be thought of, but another thing which should be borne in mind is the possibility of beginning brain tumor. In connection with the possibility of cold and rheumatism there is a case reported by Raymond (1890) of a woman, aged 25, who caught cold and had paralysis of the left lower facial, then of the 12th, of the motor 5th and 6th, and of two branches of the 3d nerve. The discs were normal. Recovery occurred in three months.

Another case is that of Michel (1872), a soldier, 23 years of age, who was attacked with pain in temples and dizziness; temperature 39.5. On the fourth day there were complete right oculomotor paralysis and hyperemia of the right disc. On the seventh day pain was felt in the knee. The eye paralysis disappeared in eleven days; the rheumatism continued for five weeks; the patient ultimately got quite well.

Of the cases of diphtheritic paralysis, one is reported by Futterer in 1896 not dissimilar in symptoms to the case under discussion; there was complete oculomotor paralysis, followed by complete recovery. There is no evidence that the patient Dr. Herrick shows has recently had diphtheria; and moreover, it is very unusual to have in diphtheritic paralysis any involvement of the sphincter of the iris. Besides, there has been no palatal paralysis in this case.

Of the influenza cases accompanied by ophthalmoplegia, one case is reported by Gutman (1890) with right ptosis and right internal and external ophthalmoplegia, and aortic insufficiency, and one still more like the case before us by Schirmer (1890), in which there was right ptosis, prominent eyeball, total right-sided ophthalmoplegia; the right pupil was rigid to light and accommodation; the eye ground was normal; vomiting, headache and diminished sensibility were present. In right half of the face were other symptoms; the tongue deviated to the right as in this case, and the masticatory muscles were weak on the right side. This case of Schirmer's is almost identical in symptomatology with the case Dr. Herrick has just presented, except that in the present case the optic discs are pale.

Of the cases of tubercular meningitis which have presented similar symptoms, one reported by Seitz (1874) had complete right oculomotor paralysis coming on suddenly, as the first sign of the disease. The autopsy eight days later showed the right pedunculus cerebri to be externally red and softened, and the right oculomotor nerve friable. Kahler (1887) had a case of complete oculomotor paralysis in a woman of 36; the eye-grounds were normal; the temperature was 38 degrees; slight stiff neck was present; later, paralysis of the 7th nerve occurred. Tubercular meningitis was found at the autopsy.

Among the recorded cases of multiple sclerosis Dr. Barker could not find a case like the one presented. There are cases of eye muscle paralysis reported, it is true, but not one at all similar to this symptom-complex which Dr. Herrick's patient presents.

Certain cases of tumor cerebri in the literature resemble closely in symptomatology this case. For instance, Sir Charles Bell had a patient with left ptosis, rigid bulb, dilated right pupil, anesthesia and neuralgia of the left side of the face, neuroparalytic keratitis, paralysis of the masticatory muscles on the left side, left eyesight poor, and temporary paralysis of the 7th nerve. The autopsy revealed a tumor in the cavernous sinus lateral to the foramen for the meningeal artery, extending forward as far as the superior orbital fissure. In this case the 3d, 4th, 5th and 6th nerves were included in the tumor, and were atrophic. The second nerve went over the tumor and was gray. It would be easy for the chiasm to be affected there. A lesion in the same situation could account for the symptoms in Dr. Herrick's case very well.

Türck (1885) had a patient, 34 years of age, who four months before death suffered violent pain in the right supraorbital region, followed the next day by right ptosis and almost complete paralysis of the right oculomotor nerve. The autopsy showed a tuberculous dura compressing the ophthalmic nerve and the right half of the chiasm, and there was infiltration of the right oculomotor.

Rosenthal (1886) had a case of left ptosis, double choked disc, partially left ophthalmoplegia, anesthesia of the trigeminus due to a tumor the size of a bean, involving the left Gasserian ganglion, oculomotor nerve and cavernous sinus.

Mingazzini reports the case of a man of 30: Left nosebleed, left tinnitus; left headache; left ptosis, and later, complete left internal and external ophthalmoplegia; left pupil wider; vision of left eye impaired; paresis of the muscles of mastication on the left side of the face, with hyperesthesia of the left side of the face and pallor of the left optic disc. Sarcoma from the antrum of Highmore, including the turbinated and the left sphenoid bone, infiltrating the sella turcica, and extending toward the clivus.

The evidence from the literature, then, is in favor of a lesion in this case of Dr. Herrick's, in the region of the cavernous sinus, probably under the optic nerve, involving the motor part of the 5th, 3d, 4th and 6th nerves there. Whether a localized meningitis (tubercular or other) or a neoplasm Dr. Barker would not attempt to say, but he thought it most likely one of these two.

In reply to a question from Dr. Williams as to whether in any of these cases there was such an acute onset, Dr. Barker said that in one the paralysis was sudden and complete at the beginning. In some of these cases the tumor developed for some time before any of the acute symptoms particularly referable to the nerves appeared, and then the nerve symptoms came suddenly. Tubercular disease is likely to produce a sudden change from the fact that it causes thrombosis of the small veins, which often results in a hemorrhage and causes the actual paralysis.

Dr. Herrick called attention to the connection of the removal of the glands from the neck in this case, which he had tried to reconcile with the acute onset and the utter absence of symptoms preceding it. There was no history of lues.

Dr. Mix asked if there would not necessarily be signs of involvement of the ophthalmic division or the superior maxillary division of the 5th, if the lesion were where Dr. Barker thinks it is. They lie in the cavernous sinus, and tumor at that point would involve the 6th nerve, the 3d, 4th and the two upper divisions of the 5th, as well as the inferior maxillary as it goes through the foramen ovale.

Dr. Barker said that the cases in the literature showed that these could escape.

Dr. Herrick said that the patient was on specific treatment now.

Dr. Billings said he had a case like this in the hospital, in which there was a left ptosis with ophthalmoplegia and fixation of the iris; the ciliary muscles seemed to be normal. She had a marked supraorbital neuralgia and great tenderness over her left temporal region, but no other involvement of the 5th. The symptoms entirely disappeared under iodides.

Dr. Moyer had seen a case almost identical with this at Bellevue, which was found to be osteosarcoma. The probabilities, he thought, were that this case was the same, because if due to specific infection it would present other features by this time. It was either tubercular or a new growth.

A Cured Case of Myasthenia Gravis was presented by Dr. Harold N. Moyer. The case had been shown to the society in October in 1902, when all had agreed that it was a true case of myasthenia gravis. Dr. Moyer had first seen the case in August of that year, when it had been sent to him by Dr. Pusey, whom the patient had consulted on account of trouble with his eyes. He could not focus the two eyes, and had drooping lids. Subsequently this weakness extended to his arms and his legs, so that he was shortly spastic all over. He was worse from the 15th of December, 1902, until the 10th of February, 1903. He could not completely dress himself during that time and was almost helpless. He could not eat solid food, as he could neither masticate nor swallow it. He could walk about the room, but could not get on his feet without assistance. His improvement, when it began, was slow, but steady. He seems now as well as ever, but has slight weakness in the fingers in the way of grasp, the dynamometer showing 15 with one hand and 30 with the other. The legs are strong and the vision is normal. The eyes did not move from the middle of August, 1902, until the middle of February, 1903. The amelioration of the symptoms began two years ago, and he has considered himself well for a year and a half. He walks from a mile to three miles every day, and could easily walk five miles a day. He can now use the typewriter, which he had been obliged to give up. He has no sensory symptoms. When the patient was presented he had the usual symptoms of coated tongue and general failure of elimination, but never any rise of temperature. He had every symptom of typhoid fever without the fever. These have all passed away. At one time during his illness he had trouble with his breathing, which he attributed to some treatment he was using. He could not get out of his chair and had to call for help. Dr. Moyer had made a diagnosis of myasthenia gravis from the symptoms when the patient was sent to him by Dr. Pusey. He had shown patellar reflexes, but these would soon exhaust, one or two strokes being all that could be elicited. He first noticed improvement in February, exercised about the house until April, when, with the assistance of a friend, he tried to get a few blocks to a barber shop, but had to stop and lean on the fence for support after going a short distance. In July he had gotten so he could go around without assistance. The treatment given by Dr. Moyer had been nitrate of strychnine.

Dr. Billings asked if it were surely myasthenia gravis, and if there were records of cases cured in so short a time.

Dr. Moyer replied that no one who had seen the case had a doubt of the diagnosis. While cases had been reported cured, none had been reported cured for so long a time. His condition was so serious during the winter that it was not expected he would live through the summer. A pretty fair reaction could be elicited for one, or may be, two jerks, and then would disappear. Asked as to the nasal smile, it was said this did not exist, but the features were mask-like, a fixed. expressionless look, and the eyes did not turn. He would look about by moving his head.

A Case of Pachymeningitis Cervicalis Hypertrophica, Simulating Syringomyelia.—This case was presented by Dr. Julius Grinker. J. W. B., aged 26, male, colored, single, electrician, came under his observation Jan. 28, 1905. His father died by accident at the age of 47; his mother died of an unknown cause at the age of 22. He was the only child. The grandparents lived to old age. No tuberculosis, carcinoma or nervous disease are found in the family record. His habits were always good. He never drank nor smoked to excess. Of venereal diseases he had a mild case of gonorrhea, but never syphilis.

The patient was well up to the age of 21, at which time he developed dysentery while serving as a volunteer in the Spanish-American war. One year later he was sick with typhoid fever and cerebrospinal meningitis. He states that during the acute stage of his illness he suffered from fever and stiffness of the neck muscles. For a period of thirty days he had pains in his back, left leg and left arm. The pains were sharp and shooting, like neuralgia. When, at the end of a month, they had entirely disappeared a period of ill-health occurred, and lasted about three months. During this time he was weak and unable to work. Within four months after the beginning of the disease he could resume his usual occupation, and boasts of having been able to carry a sewing machine up a flight of stairs. Even at this time he complained of one symptom that annoyed him since the termination of the acute stage, namely, that the sudden turning of the head would regularly cause him to become dizzy. This symptom lasted until two years ago.

About four and a half years ago, that is, about one year after his attack of typhoid fever and meningitis, he experienced a peculiar feeling in the left hand and left leg, which he described as a "tight, sleepy feeling," which was accompanied by muscular weakness, which gradually grew worse. On the left lower extremity this paresthesia involved the foot and the leg to half-way between the ankle and the knee. Five months later the right hand became similarly affected. He never experienced any abnormal sensations in the right lower extremity. One year after the onset of the paresthesia the right hand improved, but numbness continued in the left side. There was in addition a dragging sensation and weakness of the back and left leg, which was always worse when the patient walked up a flight of stairs. In September, 1901, while the patient was marching with his regiment he was unable to obey his officer's command for a "quickstep;" his feet seemed to stick to the ground.

In 1902 the muscular weakness in both hands became more pronounced. For the past two years he has been unable to raise his hands over his head, and he noticed that his shoulder girdle had become very weak.

Two years ago the patient scalded his left hand with hot water, which he did not feel, but which blistered his skin considerably. The vesicles disappeared after a time, but new crops of vesicular eruptions continued to come, which were rather slow in healing.

Bladder disturbances began very early in the disease and have continued ever since. He cannot hold his urine very well, and if he fails to empty his bladder frequently there is an overflow of its contents. Constipation and sexual indifference amounting to impotence began at the same time, although up to one and a half years ago he still had the desire, but no power. Now even his sexual libido is in abeyance.

The weakness in his hands and left leg and foot, which has been diagnosed by various physicians as hemiplegia, lead paralysis, multiple neuritis and hysteria, has of late entirely disabled him from following his occupation. Of his sensory disorder he is entirely ignorant, and it is chiefly on account of his trophic disturbance that he seeks our advice.

Status Præsens: The patient is a well-nourished boy of about 5 feet 6 inches in height and about 145 pounds in weight. His gait is somewhat spastic, but his left side is worse, resembling very much the condition of left hemiplegia. The left hand is the seat of trophic disturbance. Upon the dorsum are seen two large blebs and a generalized puffiness with edema. The third and little fingers present deep ulcerations on their dorsal surfaces, which are fully two inches long. Evidence of former vesicular or pustular eruptions are seen over various portions of the body. The face is symmetrical, the palpebral fissures are of equal size, the pupils are equal and react well to light and accommodation. There is no abnormality of the eye muscles, no nystagmus, no optic atrophy nor optic neuritis. The tongue does not deviate. The right hand shows a considerable flattening of the palm and some wasting of the extensors on the dorsum. Flexion and extension, adduction and abduction of the fingers are still possible, but below normal; in the wrist, flexion and extension are very weak. The left hand is almost paretic, but atrophies cannot be seen on account of its puffy condition. Flexion and, particularly extension of the wrist, are greatly impaired. In the left foot dorsal flexion is almost impossible; the muscles are atrophic. Although the patient considers his right foot perfectly normal, a similar weakness and atrophy of muscles is observed, which is less marked on the left side. The lower extremities appear spastic upon passive motion.

The left thenar and hypothenar eminences are rather soft and flabby. The lumbrical and intercostal muscles are extremely weak. The left triceps has almost entirely disappeared. The right triceps is present, but weak. In the right hand pronation and supination are possible, but below normal. Supination is impossible in the left hand. Wrist extension on the right side is slightly reduced; on the left side, impossible. The pectoral muscles appear atrophied in their upper portions; adduction of the extended arms can be easily prevented. The deltoids, anterior and middle portions are well developed, but the posterior portion is defective bilaterally. The upper portion of the trapezius muscle is bilaterally well developed; part of the middle and the entire lower portions are defective. The serratus magnus has completely disappeared on both sides. The paralysis becomes very marked upon an attempt to abduct the arms or raise the arms above the horizontal position, which latter is entirely impossible. When the patient attempts to extend the arms forward the scapulæ project winglike from the thorax, so that the observer can place his hands between the scapula and thorax. When the arms are extended outward the inner borders of the scapulæ touch.

An electrical examination of the weak muscles yields response to both currents, but the quantity of current required for some of the muscles is enormous. Besides the quantitative changes, it is impossible to detect either the partial or complete reaction of degeneration in any of the muscles, and it goes without saying that there is no response over muscles that have entirely disappeared. The palpable nerves are not enlarged.

Reflexes: The superficial reflexes are present and of normal intensity, with the exception of the scapulo-humeral bilaterally, and the left plantar reflex, which cannot be elicited. Of the deep reflexes, those for the upper extremities are abolished; the knee-jerks and Achilles jerk are equally exaggerated. Babinski's and Oppenheim's sign are absent. There is no tremor, but a flickering in the affected muscles can be plainly seen. There is no ataxia of station nor of gait.

Sensation: The tactile sense is involved in certain areas. The dorsum of the left hand is entirely anesthetic. Other areas are hyperesthetic; that is, the lightest touch with a wisp of cotton cannot be felt, but a touch with a coarse object, as the finger or test tube, will be perceived. The sense of position is approximately normal. The pain sense is disturbed. The temperature sense was tested by means of test tubes filled with hot and cold water. In a general way the disturbances of the temperature sense both for heat and cold are found in the same areas, although there is greater disturbance for heat than for cold.

Vasomotor disturbances are not apparent; perhaps because the patient is colored.

This corresponds in many phases with syringomyelia. The tactile sense is slightly involved, or not at all. Some will no doubt still claim that this is syringomyelia. That has the trophic disturbance, the motor disturbance, but we know since Charcot described his disease that we can get the picture from compression. That is what is now called meningomyelitis. In pachymeningitis cervicalis hypertrophica it is usually stated there is spasticity of the lower extremities, bladder involvement, rectal disturbance and sexual disturbance, but no atrophy, showing that some portion of the cord lower down must have been involved. Dr. Grinker believed that the case was one of meningomyelitis which began in 1900, went through three typical stages: a painful stage, a stage of ill health, and a last stage of ascending and descending degeneration. His reflexes are all gone in the upper extremities. He has a great deal of spasticity and a degree of ankle clonus, but not well marked. The Achilles jerk is very limited. The sphincter reflexes are present below, but not above. No Babinski and no plantar reflex are seen. The Gordon reflex is obtained, but the essayist is not sure of its reliability. He has the peculiar sensory disturbance, the trophic disturbance, pain and no history of syphilis, no tuberculosis in family, slow onset, three periods, bladder disturbance, sphincter disturbance. Dr. Grinker thought the case was one of pachymeningitis cervicalis hypertrophica simulating syringomyelia, or a case of chronic meningomyelitis. Strobe takes a decided stand against the former. He says it is not always confined to the cervical region; there is not always a hypertrophy. The author thinks many of the cases reported as syringomyelia will, on close examination, be found to be pachymeningitis cervicalis hypertrophica. The eye seems perfectly normal. The sensation of the bladder is one of fulness and the patient knows he has to pass water, consequently there cannot be anesthesia there, but there is incontinence. He never passes water if he can help it. The sphincter symptoms in connection with the others are explained by the compression of the cord below the cervical area. It is known that the fibers come from above. There is secondary degeneration below, therefore we get spasticity and exaggerated reflexes because the pyramidal tract has been involved in a myelitis. In Charcot's disease there is a period of irritation, and after that a period of degeneration, followed by anesthesia.

Dr. Barker asked as to the patient having been in the tropics. Dr. Grinker said he had been in Cuba. In leprosy the anesthesia appears in patches and never is glove shape, as in this case. In leprosy there is no exaggerated reflex. There is not a symptom of anesthetic leprosy. He has no sign of it; and then, the face is always involved.

Dr. Moyer said he saw nothing in the case inconsistent with anesthetic leprosy, as he understood it. The root symptoms, the meningitis, he had.

Dr. Grinker said that this argument was sound, but that most of the books say that in that disease the anesthesia occurs in patches.

Dr. Barker asked how the glove patches were reconciled with root lesions or cord lesions. That has been given as belonging to the segmental, and that we cannot answer.

Dr. Grinker said that in the meaning of the German word this could not be reconciled, but in the meaning of the French word "segmentaire" it was.

Myoclonus Multiplex.—This case was presented by Dr. O. M. Steffensen, who stated that the patient was a male, age 26, married; born in the United States. The family history was completely negative. The patient had had measles at six years of age and had made a good recovery. He had been engaged in prize fighting from his 17th to his 21st year. Five years ago he fell on his back from a scaffold 15 feet high, but apparently sustained no severe injuries, going back to work within a few days. Ten days after the accident he noticed tremors in the arms, legs, eyelids and various skeletal muscles. The tremors or clonic contractions were bilateral, varying in amplitude and frequency, averaging about 90 per minute and appearing intermittently. They may come spontaneously, but usually from voluntary act, such as mastication, yawning, etc. This condition was more pronounced on the left side; the sensation was perfectly normal, the reflexes somewhat increased, especially on the left side. There was no weakness of any muscles involved, neither were there fibrillary tremors or changed electrical reactions. The movements have sometimes thrown the patient from his seat. They were not present during sleep. The patient appeared mentally depressed.

PHILADELPHIA NEUROLOGICAL SOCIETY.

February 28 1905.

The President, Dr. Joseph Sailer, in the Chair.

A Case of Lumbo-thoracic Syringomyelia.—This case was exhibited by Dr. Ralph Pemberton for Dr. William G. Spiller. The patient, an Italian woman, aged 36 years, said she had always been healthy as a child, except for some eruptive fever affecting the face. She had never been to a hospital until admitted for the present complaint. She was married ten years ago and had a child eight years ago, which lived three days. She has had no miscarriage, and though she has been in America for eight years and in the hospital for the past two years, she cannot speak English.

The history of her present illness is very vague, and little can be learned beyond the fact that five years ago weakness in her legs, difficulty in walking and pain in her back developed. Her intelligence and her dialect are too poor to furnish further dependable data, even through an interpreter.

The pupils are slightly unequal, the right being larger than the left, but they both react promptly to light and in accommodation and convergence. The vertebral column shows no deformity.

The motion, coördination and sensation of the arms and hands are good. The reflexes are about normal in intensity, the left biceps jerk being, perhaps, slightly increased.

The legs are well developed and show no trophic disturbance in any part. In the right leg and thigh voluntary power is about normal, but in the left leg and thing it is distinctly diminished, though not lost. On the left side are also a greatly increased patellar reflex, distinct patellar clonus, persistent ankle clonus and a typical Babinski sign. On the right side the patellar reflex and the Achilles jerk are completely lost, but Babinski's sign is distinctly present, though not quite so pronounced as upon the left. Over both legs light touch is promptly perceived, but on the right side pain sensation is undoubtedly diminished, though pin prick is felt if the prick be deep. Over the whole right lower limb sensation for heat is much diminished, and also apparently over the lower part of the abdomen on the right side, but over the left leg and thigh perception of heat and pain is very prompt. Sensation of cold probably is also diminished in the entire right lower limb.

The gait of the patient is peculiar, as owing to the spasticity of the left leg, she swings the leg somewhat from the hip and scrapes the toes of the left foot along the ground. The right lower limb is thrown somewhat unnecessarily forward in walking and the heel is brought down before the ball of the foot, and the foot describes a small arc outward.

Some spasticity develops on passive motion of the left leg, but the right leg is entirely flaccid and the heel-to-knee movement, which cannot be performed on the left side on account of weakness, is easily and coordinately performed upon the right side. There is no ataxia of station.

The general health is good. She has no disturbance of bladder or rectal functions, though two years ago she developed incontinence of urine for a short time. Her temperature, pulse and respiration are normal.

The Babinski Sign Occurring in Strychnine Poisoning and Acute Lead Encephalopathy.—This report was made by Dr. Ralph Pemberton.

Case I.—A white female, age 30 years, with diagnosis of melancholia and possibly petit mal, took between 1½ and 2 grains of strychnine sulphate. Pain and rigidity in the feet and legs developed in about ten minutes, with intermittent convulsions, which grew more frequent, and finally became general. Bilateral ankle clonus was present at the end of the first ten minutes of the attack, with a plain and prompt Babinski sign on the right side. Urgent need of treatment prevented testing for it on the left side. Babinski's sign and ankle clonus had not been present before the attack, and were not present after an uneventful recovery, though the patellar reflexes were somewhat increased.

Case II.—A colored male, aged 23 years, presented a history of working in lead for nine months, and in that time had had two attacks of malaise, abdominal pains, nausea and vomiting. The second attack was accompanied by unconsciousness.

The patient was seen about 34 hours after the onset of a third attack, which was similar to the second except that the third was more severe. He was then in pronounced stupor, could be roused with great difficulty to mutter inarticulately, and presented a plain Babinski sign on the left and a doubtful one on the right side. The reflexes elsewhere in the legs were depressed. On the following day Babinski's sign was still present, and could also be elicited on the left side by stroking the sole of the right foot; *i. e.*, it was a crossed Babinski phenomenon. On the next day the Babinski sign had entirely disappeared. The patient was by this time almost clear mentally, and made an uneventful recovery. The diagnosis was lead encephalopathy.

(Case I) Progressive Muscular Dystrophy, with Atrophy of Bone; (Case II) Chronic Mercurial Poisoning, and (Case III) Tabetic Facial Palsy.—These cases were exhibited by Dr. William G. Spiller.

Case I.—Muscular dystrophy had existed since the second year of life. The man had had no acute disease and never any pain. Sensation was not affected. He had marked atrophy of all parts of the body, and the atrophy had progressed gradually and had become intense, and was asymmetrical in some parts. Dr. Spiller thought this was not a case of poliomyelitis because of the history. The humerus on the left side was markedly smaller than that on the right side.

Case II showed a rare condition of chronic mercurial poisoning. There was marked tremor, increased by active movement. At times this tremor was present in all parts of the body, even in the soft palate and larynx. The larynx was congested, suggesting disturbance of the liver. Degenerative changes were seen in the optic discs. The speech was tremulous.

Case III.—This was one of tabes in which the diagnosis was made because of loss of the patellar tendon reflexes, loss of the Achilles tendon reflexes, occasional shooting pains in the legs, some difficulty in holding the urine, ptosis of the left upper eyelid, paresis of the left external rectus muscle, paralysis of the right external rectus muscle, irregularity of the pupils, Argyll-Robertson pupils and optic atrophy. In addition the man had paresis of the right side of the face, of which he was entirely ignorant.

Dr. Dercum thought Case I was remarkably interesting, inasmuch as the man had changes not only in the limbs, but in the bones of the face, the right malar bone being smaller and the zygomatic arch narrower than the corresponding structures on the left side. He thought there was something in the face to suggest hemifacial atrophy, while the limbs called to mind some of the appearances of morphea. No one has ever explained the relation between morphea and these deep-seated dystrophies. That there is some relation is extremely probable. We are entirely in the dark with regard to their pathogenesis. The only autopsy of a facial hemiatrophy which Dr. Dercum could recall was that of Mendel, made many years ago. Mendel found a degeneration of the descending root of the fifth nerve. In the present case there are no skin changes such as we are apt to find in hemifacial atrophy. Dr. Dercum thought the case went far to prove the kinship between atrophic and dystrophic diseases.

Dr. Lloyd stated that this case recalled to his mind a case he reported to this Society some years ago of pseudo-hypertrophic muscular paralysis with trophic changes in the joints. That case was of the pseudo-hypertrophic type and not the atrophic type. The changes were principally in the elbows and knees.

Dr. Mills referred to a case he had reported several years ago where the symptoms were very similar to those of Dr. Spiller's Case II. The patient had worked in the same factory where Dr. Spiller's patient was employed. His case had some of the features of general paralysis, notably tremor, peculiar tremulous speech and a depressed mental condition.

Dr. Dercum thought it very remarkable that when the poisoning by mercury takes place through the respiratory tract the symptoms are so different from those present in poisoning caused by hypodermic administration, by inunction or by internal administration. The question arises whether we have not a mixed intoxication to deal with, *i. e.*, one due not solely to mercury. It is interesting, too, to note that many of the symptoms resemble those of chronic alcoholic poisoning.

In connection with Dr. Spiller's third case (tabetic facial palsy) Dr. Gordon mentioned a case he saw several years ago of tabes in which facial palsy occurred six months after infection. The symptoms of tabes made their appearance one year after the infection. The facial palsy was not a paresis, but a true Bell's palsy. After the patient was cured of palsy the tabetic symptoms became much aggravated.

Dr. Spiller, in closing the discussion on his cases, stated that in 1898 he had reported a case of muscular dystrophy with involvement of bone. In regard to the man with chronic mercurial poisoning, he said the gums were much affected.

Hemorrhage in the Floor of the Aqueduct of Sylvius.—Dr. Weisenburg exhibited a patient with hemorrhage into the nuclei of the third and fourth nerves. This patient was seen with Dr. M. Radcliffe in the Wills Eye Hospital. The man, 53 years of age, a farmer, while pitching hay suddenly felt a "gripping feeling" in both eyes which was followed almost immediately by ptosis of both eyelids. This has persisted. He had never been seriously sick previously and had no venereal history. A slight frontal headache with some vertigo was present occasionally both before and since the attack, but no other symptoms were complained of.

With the face at rest there was complete ptosis of both eyelids, it being possible to see the cornea for about 1-16 of an inch. When he was looking upwards forcibly the cornea could be seen for 3-16 of an inch in the right eye and 2-16 in the left. The eyelids could be shut forcibly and well on each side. Dr. Radcliffe stated that vision in O. D. was 20-30, in O. S. 20-40. The right pupil was oval and 4.5 mm. in width, the left round and 4.5 mm. in width. No reaction to light, accommodation and convergence could be obtained. There was complete inability to move the eyeballs upward, downward or inward; external rotation being possible on each side, but weak. The ophthalmoscopic examination showed the discs to be pale, but the fundi were otherwise normal.

All of the other cranial nerves were normal. Hearing and taste were not affected. All the limbs were normal in motion, the reflexes were not altered, and sensation was not disturbed.

The sudden onset without any previous symptoms of any kind suggested most probably a diagnosis of hemorrhage in the nuclei of the third and fourth nerves.

Hemorrhage in the Pons.—Dr. Weisenburg also exhibited a patient who had a fracture of the base of the skull, which probably caused a hemorrhage in the right side of the pons. This patient was studied in the Polyclinic Hospital, in the service of Dr. Spiller. Two years ago this man, who is 48 years old, fell from the back of a trolley car and was said to have struck the back of his head. He was taken to a hospital in an unconscious condition and remained so for four days. A diagnosis of a fracture of the base of the skull was made. When he regained consciousness he was found to be paretic on the left side of the body, and he saw double. He was able to go home three weeks after the accident, at which time the paresis of his left side had almost completely disappeared. He has complained ever since of a feeling of numbness in the left forearm and of a "chilly" feeling in his left leg. He has never had headache, nausea, vomiting or convulsions, but his memory has become poor. The diplopia has persisted.

Examination showed the pupils to be of equal size. The response to light, accommodation and convergence was normal. The right external rectus was paretic. The eyegrounds were normal. The right seventh nerve was weak, but this could be attributed, possibly, to the paralysis of the sensory part of the fifth nerve, and was a sensory paralysis. The left seventh nerve was normal. The man complained of a constant roaring in his right ear, though hearing was normal. The masseter on the right side did not contract as well as it should, and a constant fine fibrillary tremor in the temporal and masseter muscles was present. There was a complete anesthesia for touch and pain in the upper distribution of the right fifth nerve and a hyperesthesia in the middle and lower distribution. There was no keratitis neuroparalytica. The patient complained of a constant feeling of heaviness in the right side of his face, this being due to involvement of the fifth nerve.

The tongue, when protruded, deviated to the right, and constant fine fibrillary tremors were present in both sides, more marked on the right. There was also a suggestion of atrophy on either side of the tongue. The sense of smell was preserved. Lachrymation was present on irritation of the left nostril, and not on the right side. Both the upper and lower limbs were normal in power and movement. Sensation was preserved in all parts of the body. All of the tendon reflexes were exaggerated and unequally so. There was no Babinski reflex and no ataxia. The bladder and rectal functions were normal.

A hemorrhage in the right side of the pons would best explain the symptoms in this case, for a lesion in this area would involve the fifth, sixth, seventh and possibly the eighth nerves, all of which were implicated in this case. The irritative sensory phenomena present in the left side of the body were probably caused by a lesion of the median fillet, a so-called central pain. Such cases are rare, but are to be found in the literature. The early hemiplegia was merely a transient symptom, and due, no doubt, to involvement of the pyramidal fibers. The bilateral involvement of the hypoglossus nerve can be explained by an extension of the hemorrhage into the nuclei of these nerves.

Dr. Dercum asked Dr. Weisenburg why he did not call the first case one of polioencephalitis superior. The symptoms of this disease may come on very quickly. Why call it hemorrhage?

Dr. McCarthy stated that he had the same thought as Dr. Dercum about this case. If the entire third nerve were involved on both sides it would necessitate a rather extensive and bilateral hemorrhage, and he thought it would be more consistent to make a diagnosis of polioencephalitis superior.

Dr. Spiller said that the symptoms of polioencephalitis superior might come on within a short time, but he thought it would be difficult to say in regard to this case whether the symptoms were due to polioencephalitis superior or to hemorrhage. He thought, however, that the distinction was not very important, as in cases of polioencephalitis often there are small hemorrhages, and a lesion need not be very great to destroy the oculomotor nucleus. It would have to be a bilateral hemorrhage, of course, but he had seen hemorrhage in the tegmentum of the pons go beyond the raphe and extend to the other side.

Dr. McCarthy stated that he had specimens of polioencephalitis superior which showed miliary hemorrhages and marked degeneration of the nuclear cells. The miliary points of hemorrhage were like what is seen in the anterior horn cells in anterior poliomyelitis.

Dr. Weisenburg, in closing the discussion on his case of hemorrhage in the floor of the aqueduct Sylvius, stated that it would be rather difficult to make a diagnosis between superior polioencephalitis of Wernicke and hemorrhage, but in a man who had had no symptoms at all and in a moment bilateral ptosis developed one would naturally look for hemorrhage, rather than polioencephalitis.

A Case of Malarial Infection Presenting Symptoms of Multiple Neuritis.—This report was read by Dr. George E. Price.

Dr. Weisenburg stated that while in the Philippines he saw a great deal of malaria, and a great many nervous symptoms were present in those cases. He mentioned the case of a surgeon who became suddenly weak in both knees and fell. He was taken to the hospital and found to have all the symptoms of multiple neuritis, due to malaria. He stated that he had seen perhaps six cases of malarial neuritis in the two years he was there. So far as treatment was concerned, quinine was given in massive doses. Occasionally a vein was opened and .50 or 60 grains injected.

Clinical and Pathological Report of a Case of Lead Poisoning, with Remarks on the Pathogenesis of the Disease.—This paper was read by Dr. Alfred Gordon.

Dr. Dercum thought the interesting point in this report was the posterior sclerosis. Posterior sclerosis as a result of chronic lead poisoning is a novel finding. It is most suggestive as to the action of poisons in general.

Periscope

Archives de Neurologie

(Vol. 19, 1905, No. 1, January.)

1. Peripheral Facial Paralysis Due to a Fibro-Sarcoma Enveloping the Nerve at its Point of Departure from the Bulb. Raymond, Huet and Alquier.
2. Therapeutic Notes on Veronal in the Insane. Serieux and Mignot.
3. Sclerotic Atrophy of Left Hemisphere, Imbecility, Right Hemiplegia, Epilepsy, Vertigo, Dementia. Bourneville et Mangeret.

1. *Peripheral Facial Paralysis.*—This case is of interest, as it is rare for a tumor occupying the lateral part of the bulb to give rise to the syndrome of peripheral facial palsy only; also because of the origin and nature of the tumor. The palsy was of thirteen years' duration; there was no involvement of sensation; it was of peripheral type, with marked R. D. For a term of five years it was stationary and no other nerve was involved. Autopsy showed broncho-pneumonia as the cause of death. A globular tumor was found on the left side, situated between the bulb and the cerebellum, so disposed as to involve only the 7th, leaving the other cranial nerves and the pyramid quite uninvolved. (1) The tumor was found to be an encapsulated fibro-sarcoma; (2) the facial nerve-fibers showed a marked atrophy and degeneration; (3) Gasserian ganglion of the left side contained a greater number of pigmented ganglion cells with chromatolysis than on the right side; (4) the small branches of the facial, orbicularis, zygomatic major, orbicularis oris, chin muscles were affected; (5) the bulb showed no alteration, except simple atrophy of the cells of the 7th nucleus. The diagnosis was not possible during life, as the whole syndrome was precisely that of ordinary facial palsy. The classification made by the authors is that of a neuro-fibro-sarcomatosis as described by Cestan, limited, however, to one nerve root, sparing all other tissues, so that this palsy of thirteen years' duration presented no other symptom which could aid in localization.

2. *Therapeutic Notes on Veronal in the Insane.*—The result of careful observation on twenty cases is given. The cases were not picked cases, but were mostly those of old men and women. The drug was given in cachets with a hot drink, or in suspension in milk or water, or even mixed with the food. *Melancholia.*—Its action was quite remarkable. In doses of 0.3 to 0.5 it was superior to all other hypnotics. Sleep ensued in one or two hours, and was usually calm and sufficiently long. In spite of long continuance, there was no habituation. In addition to the excellent hypnotic effect it produced a diminution in the agitation and anxiety, but was without influence on the delirium or depression. *Dementia Præcox.*—It was usually as satisfactory here, except in one catatonic case. *Paresis.*—Here the action was less satisfactory, inferior to the bromides and prolonged baths. Sleep came on only after four or five hours and was of short duration. *Conclusions.*—In melancholia and agitated dementia veronal is an excellent hypnotic. Sleep comes on usually in from one to two hours and lasts several hours. Its action is more marked after several days' usage. There is no habituation, and it does not lose its good effect easily. The dose is from 0.3 to 1.0, never above the latter mark. It exerts as well a decided calmative influence in this class of psychoses. Its action in general

paralysis (agitated and hallucinatory forms) is not satisfactory. Sleep comes slowly, is irregular, of short duration. In depressed and simple demented forms of paresis the authors have not had sufficient experience. Even in doses of 1.0 they never found disagreeable symptoms which could be attributed to the drug. There was no modification of the pulse, no alteration of cardiac murmurs; old people, even those with cardiac lesion, bore the drug well. Albuminuria was *never* observed. In only one case out of forty was there a marked intolerance. The drug was often given for long periods night after night. They regard is as the most serviceable of hypnotics.

3. *Sclerotic Atrophy of Left Hemisphere.*—On account of the remarkable accuracy in observation extending over years and including the parents, as well as the very careful autopsy, study of this case is of interest. The points are the first convulsions at six months in an infant supposedly normal, followed by a transitory hemiparesis (right). At ten months a second period of illness occurred with similar convulsions and paresis, with respite until the fourteenth month. After this convulsions every six months until the second year. After about the seventh year the fits became more frequent, and finally, at about eight years occurred daily. Intelligence diminished, and finally dementia ensued. The character upon admittance (eight years and four weeks) was noticeably expansive, but became more and more suspicious, irritable and violent.. The authors note that the ability to calculate and write disappeared before that of reading, which also finally went.

Autopsy showed generalized tuberculosis of both lungs and the kidneys. Skull: Persistence of the sutures, great thickening of the bones, left side. Encephalon marked difference in weight of the two hemispheres, the left 200.0 less. General atrophy left cerebrum, convolutions pale, indurated, thinner; atrophy most marked over the frontal and occipital lobes. Also on the left side the optic nerve, the optic radiation, the mammillary body, the cerebral peduncle, the corpus striatum and the thalamus were all diminished in volume. Besides, the left hemisphere presented a chronic meningitis. The cerebellum was also smaller. Changes in the right brain were of small moment. There was a partial epilepsy of hemiplegic type due to the sclerotic atrophy and the chronic meningitis of the left hemisphere. The thickening of the skull on the left side was a sort of compensatory process following atrophy of the left hemisphere. There was dilatation of the left lateral ventricle without increase of fluid. The authors conclude that the hemiplegic type of epilepsy with meningo-encephalitis or with chronic meningitis is in every way similar to that form of epilepsy which terminates in dementia.

WOLFSTEIN (Cincinnati).

American Journal of Insanity

(Vol. 61, 1905, No. 3.)

1. Fifty Years in Psychiatry. J. B. CHAPIN.
2. Calcification of Finer Cerebral Vessels, with Remarks on Its Clinical Significance. A. PICK.
3. On the Methods of Later Psychiatry. CLARENCE B. FARRAR.
4. Remarks upon Insanity and Epilepsy in Regard to Duration of Life. ROBERT JONES.
5. A Case of Glioma of the Pineal Region. E. E. SOUTHARD.
6. Some Observations on the Progress of Psychiatry. T. R. NICHOLS.
7. The Question of Dementia Præcox in France. A. V. PARANT.

1. *Fifty Years in Psychiatry.*—The address delivered by the author at a dinner given to him by his friends on the occasion of the fiftieth

anniversary of his entrance upon work in hospitals for the insane. The changes which have occurred and the progress which he has seen during his long and useful life are interestingly described.

2. *Calcification of the Finer Cerebral Vessels.*—As long ago as 1894 the author called attention to a calcification of the smaller cerebral vessels of non-atheromatous nature which he had observed in comparatively young subjects. Taking up the subject again after maturely considering the meaning of his findings, he discusses at some length the anatomical characteristics of the changes found in the calcified vessels, and goes over the histories of two cases, respectively those of a man of 24 and a woman of 42 years of age in whom, in conjunction with gradually increasing mental dullness and eventually stupor, there were convulsive attacks with greatly increased myotatic irritability, and Trousseau's and Chvostek's signs, giving the clinical picture of tetany. In each of these cases there was marked calcification of the smaller arteries of the brain. While not claiming that these changes constitute the pathological basis of tetany, the author holds that they have an important bearing upon the subject.

3. *On the Methods of Later Psychiatry.*—An interesting review of progress in psychiatry, in which the contributions of the clinical, the anatomo-pathological and chemical, and the psychological methods are successively considered. The author urges that a greater number of carefully selected men be placed upon asylum staffs, and thinks that cut-and-dried methods of classification are to be avoided, patients being studied as individuals by all the methods at our disposal. All these methods, however, cannot be concentrated in the hands of any one individual, and for any real progress the coöperation of the clinician, the psychologist, the pathologist and the physiological chemist is indispensable.

4. *Insanity and Epilepsy in Regard to Duration of Life.*—A discussion of the bearing of insanity and epilepsy upon the question of life insurance. The following conclusions are drawn: (1) Insanity *per se* is inimical to life. Expectation of life is least affected in paranoia and in other cases having fixed delusions; also in cases of moderate, but chronic, weak-mindedness after acute insanity. Congenital weak-mindedness diminishes the expectation of life. (2) The most powerful and the most frequent antecedent of insanity and of epilepsy is either ancestral insanity or epilepsy. (3) Medical forms used in insurance offices should include an inquiry as to insanity in ascendants and collaterals. (4) Suicidal tendency is entirely hereditable. (5) Suicides are only half as frequent in asylums as among the general population during the period of greatest liability. (6) Phthisis and insanity are features strongly convergent toward insanity. 7. Epilepsy shortens life more than insanity. (8) Antecedent syphilis cannot always be ascertained in cases applying for life insurance, and general paresis occurs in not more than 1 per cent. of all cases having had syphilis.

5. *Glioma of the Pineal Region.*—A case of tumor of the pineal gland in a man 44 years of age, whose symptoms consisted of disorientation, defective memory and general deficiency, with inability to stand alone, weakness of the legs, with dragging of the left leg, tremor of the hands, increased knee jerks and sluggish pupils. Death from lobar pneumonia. Nephritis and arteriosclerosis were also found. The tumor springing from the pineal gland encroached upon the third ventricle and aqueduct of Sylvius. Microscopical examination showed it to be made up of cells and glia tissue, with numerous calcareous deposits, hence, while it may be called a psammoma, the author thinks that in strictness it belongs to the class of gliomata.

6. *Progress of Psychiatry.*—A paper read before the Medical Association of Texas giving in outline the progress made in psychiatry in

recent years, with suggestions as to further improvement in the care of the insane.

7. *Dementia Præcox in France.*—In the form of a letter from France, the author discusses the views which at present prevail among French alienists with regard to this disease. While a few have accepted the views of Kraepelin in their entirety, there appears among the majority a hesitation about recognizing the dementia præcox of the Heidelberg school as a definite clinical entity. A useful bibliography is appended.

ALLEN (Trenton).

University of Pennsylvania Medical Bulletin

(January, 1905.)

1. General or Localized Hypotonia of the Muscles in Childhood (Myotonia Congenita). WILLIAM G. SPILLER.
2. Congenital Spastic Rigidity of the Limbs (Congenital Hypertonia, Little's Disease). WILLIAM G. SPILLER.
3. Pseudobulbar Palsy. Report of Three Cases with Necropsy and of Three Cases without Necropsy. T. H. WEISENBURG.
4. Muscular Atrophy, Degeneration of the Trigeminal Nerve and of the Lateral Columns, and Anemic Changes in the Spinal Cord Occurring in Tabes Dorsalis. C. D. CAMP.
5. Combined Pseudo-systemic Disease, with Special Reference to Annular Degeneration. A. R. ALLEN.
6. A Pathological Study of Acute Myelitis. J. H. W. RHEIN.
7. Bulbar Symptoms Occurring with Carcinoma of Parts Other than the Central Nervous System, and Resulting from Intoxication. T. H. WEISENBURG.
8. Fibrous Nodules in the Cerebral Pia-arachnoid Causing the Appearance of Tuberculous Meningitis. C. D. CAMP.
9. Primary Degeneration of the Pyramidal Tracts; a Study of Eight Cases with Necropsy. WILLIAM G. SPILLER.

1. *Myotonia Congenita.*—The condition of congenital myotonia, described by Oppenheim, is a hypotonia of the limbs, especially of the lower, with weakness or loss of the tendon reflexes, observed within the first months of life. The electrical examinations have shown quantitative alteration, even complete disappearance of the reactions. Intelligence, sensation and the special senses are not disturbed. The condition is always congenital. No necropsy has hitherto been reported, but a case with necropsy is now recorded by Dr. Spiller. The parents of the child were living and well, and there was no hereditary tendency to the disease. One brother and three sisters were in good health. The patient was born six years after the previous child, and at full term, and was breast-fed until twenty-two months old. He had always been obstinately constipated, probably from atony of the abdominal walls. His weakness had not progressed. He was fairly well developed. The muscles were not wasted and the limbs were of good size, but the flesh was soft. The limbs were moved voluntarily, but the movements were weak. The patellar reflex and Achilles tendon reflex were not obtained on either side. Sensation to pinprick was preserved. The hypotonicity of all the limbs, especially of the lower, was very great. Either lower limb could be flexed so that the front part of the thigh and leg could be placed in close contact with the trunk, and the foot be placed behind the head without causing discomfort to the child. He could sit alone only for a minute or two, and only when balanced. He had never been able to stand. He died at the age of 22½ months.

The hypotonicity of the limbs twenty hours after death was as great as during life, and the absence of post-mortem rigidity was striking. In cutting into the muscles of the right calf the fat was found to be 6 mm. in thickness, there was very little muscle, and it appeared paler than normal. There was hardly any muscle in the sole of the left foot where an incision was made in this part, and only fat with a little muscle tissue could be removed for examination. The subcutaneous fat over the triceps muscle was 3 mm. in thickness. The microscopical study showed that muscles from the sole of the left foot, from the back of the trunk and from the left calf, had a hyaloid appearance, and those from the sole of the foot contained a large amount of fatty connective tissue and a considerable increase in the nuclei of the connective tissue. The muscle fibers were small, and those from the sole of the foot and from the calf were much smaller than those from the back of the trunk. The transverse striations were well preserved, but the longitudinal were not so distinct. The peripheral nerves, brain and spinal cord were normal.

The examination in this first case of congenital myotonia with necropsy shows that the condition is a muscular disorder, and not a disease of the nervous system. (Author's abstract.)

2. *Little's Disease.*—Four cases of congenital spasticity of the limbs with necropsy and without gross lesions are reported; two of these have been recorded previously. In Case I the brain and spinal cord were small, the Betz cells of the paracentral lobule and the nerve cells of the anterior horns of the spinal cord were not numerous, and the nerve fibers of the crossed pyramidal tracts were exceedingly fine. There was evidently an arrest in the development of these fibers.

In Case II no lesions explanatory of the spasticity were observed.

In Case III an arrest in the development of the pyramidal fibers was found, similar to that the author described in 1898. The child was born at the seventh month, and weighed 3½ pounds at birth. Convulsions began when she was a year and two or three months old, and later became very frequent. She did not learn to speak. Her limbs were very spastic, and her mental development was very imperfect. She died when two years and six months old. The nerve fibers in the crossed pyramidal tracts of the cervical region were very fine, and finer than those of the columns of Goll and finer than the fibers of the crossed pyramidal tracts from a normal child of the same age. The brain was well developed.

Case IV. The patient, a woman, was 70 years of age. Mentality was feeble. The upper and lower limbs were very spastic and contractured, and the patellar reflexes were exaggerated and Babinski's sign was present on each side. Speech was difficult. The condition was said by the patient to have existed since birth. The bodies of three or four cervical vertebræ extended backward into the spinal canal and compressed the spinal cord partially. The cervical cord in the compressed portion was considerably smaller than in the thoracic region, and the posterior columns in the cervical region were almost destroyed. The right crossed pyramidal tract was degenerated in its posterior portion, but the left crossed pyramidal tract did not present much degeneration. The right and left tracts of Gowers and the right direct cerebellar tract were degenerated in the cervical region. The case was one of the rare examples of early spasticity of all four limbs caused by a lesion of the spinal cord in the cervical region. The cause of displacement of the vertebræ could not be determined. It may have been produced by pulling on the head during childbirth. The condition of the posterior columns indicated that the lesion developed early in life. (Author's abstract.)

3. *Pseudo-bulbar Palsy.*—This is the first extensive paper upon this subject attempted in this country. One clinical and one pathological case had been reported previously in this country and England, respectively. After detailing the records of the cases and giving a résumé of findings, the clinical symptoms and the pathological findings of this disease are given in detail. The following conclusions are arrived at concerning the path-

ology: "We can come to the conclusion, therefore, that pseudo-bulbar palsy in the adult is probably always caused by bilateral lesions, and that these lesions may be in the cortex, subcortex, internal capsule, central ganglia, as the caudate nucleus, optic thalamus, especially the lenticular nucleus, cerebral peduncles, pons or medulla oblongata. The lesions consist either of areas of softening or hemorrhages which can be seen macroscopically, or may not be found unless a careful microscopic examination is made. The central ganglia, especially the lenticular nuclei, are more often involved than the other parts of the brain. It is impossible to make a pathological distinction between a purely cerebral and a cerebro-pontile type unless a thorough microscopic examination has been made, and it so it will be found in most cases that there are diffuse lesions throughout the brain, and probably also in the medulla oblongata and pons. The cause of the whole symptom-complex is usually an arteriosclerosis of the bloodvessels of the brain.

It is probable that in the cases of pseudo-bbulbar palsy reported as caused by a unilateral lesion a more careful microscopic examination of the brain would have shown bilateral involvement.

"In infantile pseudo-bulbar palsy the cause is mostly congenital maldevelopment, although cerebral thrombosis and bilateral tumors in the basal ganglia may cause the symptom-complex of pseudo-bulbar palsy." (Author's abstract.)

4. *Muscular Atrophy, etc., in Tabes.*—In the first case the patient presented the symptoms of advanced tabes complicated by muscular atrophy in the lower extremities; an intense anemia. possibly due to a suppurative process, and trophic changes in the fifth nerve, as shown by painless falling out of the teeth. Pathologically the case presented the lesions of advanced tabes dorsalis in the posterior columns of the spinal cord and in the posterior roots; marked degenerative changes in the nerve cells of the anterior horns of the spinal cord, viz., chromatolysis, displacement of the nûcleus and loss of dendritic processes; beginning changes in the cord produced by the anemia. shown by the swollen axis cylinders in the anterior columns and partial degeneration of the fifth nerve.

In the second case there was, in addition to typical lesions of tabes, a degeneration of irregular distribution in the lateral columns, probably due to interference with the blood supply. This case is interesting as affording an explanation for the return, or even exaggeration, of the knee-jerks in a case of tabes in which the posterior roots are only partially degenerated. Another important feature of the second case, especially in connection with the first case, was the correspondence of the healthy nerve cells with the unaltered musculature. From these cases and a study of the literature it would seem plain that the atrophy occurring in tabes is really due to degeneration of the anterior horn cells.

In most cases the spinal cord changes occur in pernicious anemia and the alteration is very intense, consisting of many foci of alteration; these causing secondary degeneration. From a study of this case it can be concluded that the earliest alteration of anemia, as shown by the spinal cord, consists of swelling of the axis cylinders. (Author's abstract.)

5. *Combined Pseudo-systemic Disease.*—After stating that the appearance of annular degeneration of the spinal cord can be caused by error in staining and preparing the specimen, this paper takes up the anatomy of the blood supply of the spinal cord. A case is then stated in detail of a girl afflicted with a paraplegia of spastic type and a discussion of the pathological findings is given. There was an unbroken ring of degeneration around the periphery of the spinal cord in the cervical and lumbar regions, and likewise a secondary degeneration of the columns of Goll in the cervical region. In addition was found a profuse small round cell infiltration of the pia, which in several instances could be traced entering the substance of the cord in company with the centripetal vessels.

The vascular origin of the lesions was upheld, and the monograph on the spinal cord by R. T. Williamson discussed. (Author's abstract.)

6. *Acute Myelitis.*—Dr. Rhein reports two cases of acute myelitis with post-mortem findings. Case I was a woman of 29, who developed in the fifth month of pregnancy motor and sensory paralysis of the legs, and incontinence of urine and feces. She complained of pains distributed generally over the body, but which were especially severe across the upper part of the chest. Examination of the specimens showed the presence of an acute inflammatory myelitis.

Case II was that of a woman of 39, who upon examination was found to be totally paraplegic. She was also anesthetic below the sixth rib in front and over a level four inches lower in the back. There was paralysis of the bladder and rectum. The knee-jerks were absent, but the Babinski sign was present. She died in thirty-two days after the onset of the symptoms, the paralysis having extended to the arms four days before death. There was no inflammatory change found in the spinal cord, but in the anterior horns of the thoracic and lumbar regions the nerve cells were altered.

The literature of the subject was examined and fifty-two cases of so-called acute myelitis with autopsy cited. Dr. Rhein concludes that (1) the most frequent pathological findings in acute myelitis consist of necrotic foci, thickened blood vessels, round-cell infiltration, small hemorrhages, destruction of the nerve fibers in the neighborhood of the diseased blood vessels, alteration of the nerve cells, and the presence of granular cells; (2) more rarely there may be degeneration of the nerve elements without evidences of inflammation; (3) there may be cases in which these two processes seem to be associated; (4) that infection plays an important rôle in the production of acute myelitis; (5) that the cause of acute myelitis may be either a micro-organism, the products of a micro-organism, or a toxin. (Author's abstract.)

7. *Bulbar Symptoms Occurring with Carcinoma.*—Bulbar symptoms have been reported previously only by Bruns and by Spiller and Weisenburg. In this present case a woman, aged 59 years, had had carcinoma of the breast for several years. Five months before her death she began to have bulbar symptoms, such as difficulty in talking and swallowing, and later talking and swallowing became almost impossible. The microscopical examination showed a disease of the nerve cells of the nuclei of the 6th, 7th, 9th and 10th cranial nerves. The symptoms were undoubtedly toxic in origin, and were due to the carcinoma poison. The views of Hudoverning that the alterations in certain nuclei in the bulb are the result of so-called "reactions at distance" is disputed. These changes are probably part of a general intoxication. (Author's abstract.)

8. *Fibrous Nodules in the Cerebral Pia Arachnoid.*—The patient was a male, 64 years of age, with a history of excessive alcoholism. At autopsy he was found to have a large retro-peritoneal sarcoma. There were no cerebral symptoms, but the cerebral pia was studded with numerous small white nodules, supposed to be sarcomatous on account of the coincident abdominal growth. The nodules were opaque, and resembled very closely both in appearance and distribution those occurring in tuberculous meningitis, so closely, indeed, that a differential diagnosis by the unaided eye would have been difficult. Microscopically, however, they were very different from tubercles, being composed of a fine, loosely-meshed connective tissue, poor in cells, and therefore resembling closely the Pacchionian bodies, except that they were situated within the pia and had no peduncle. (Author's abstract.)

9. *Primary Degeneration of the Pyramidal Tracts.*—The author has had the opportunity to study eight cases of primary degeneration of the central motor tracts, with necropsy. In six of these cases degeneration of the nerve cells of the anterior horns was present, so that these were

cases of amyotrophic lateral sclerosis. Two of the eight cases were ascending and at first unilateral in type, and later the paralysis became a triplegia. One began as a hemiplegia, and later the other side of the body became affected. Two may be regarded as uncomplicated cases of primary degeneration of the pyramidal tracts without implication of the nerve cells of the anterior horns of the spinal cord, and in neither of these cases was muscular atrophy present. In one case only could degeneration be traced as high as the motor cortex, but in two it could be traced into the internal capsule, and in two into the cerebral peduncle. In three of the cases the degeneration extended as high as the pons and not beyond. One of the cases showed that great spasticity of the limbs may be caused by a degeneration of the pyramidal tracts that is barely detectable, and that where a limb is spastic from such slight degeneration disease of the cells of the anterior horn on the same side as the degeneration may assist in the formation of contracture by increasing the paralysis. Some of the cases showed that disturbance of the bladder and rectum may be caused by degeneration of the central motor tracts, even where the posterior columns and the nerve cells of the anterior horns are not diseased; and that degeneration of the posterior columns is not to be diagnosed simply because this sign exists.

Dr. Spiller thinks it is questionable whether in amyotrophic lateral sclerosis the process is ascending. It is possible that there is a gradual death of the portions of the central motor neurones most remote from the cells of origin, and that the extent of the nerve fibers diseased varies in different cases, but that the whole portion of each fiber that is affected is diseased usually almost simultaneously, although the individual nerve fibers become affected at different periods.

One of the cases belonged to the type of primary uncomplicated unilateral degeneration of the pyramidal tract, although later the other pyramidal tract became affected; another belonged at first to the type of unilateral amyotrophic lateral sclerosis. (Author's abstract.)

Revue de Psychiatrie et de Psychologie Experimentale

(May, 1905.)

1. The Etiological Rôle of Syphilis in the Psychoses. MARCHAND.
2. The Curve of Intellectual Work After Kraepelin. VASCHIDE.
3. Attempted Homicide Committed by a General Paralytic with Melancholic Tendencies. DAMAYE.

1. *Syphilis in the Psychoses.*—The author concludes as follows: Syphilis by its toxin may determine the appearance of psychoses in predisposed subjects. This etiology of psychoses is rare. The mental malady appears almost always in the months following the infection. Specific cutaneous lesions coexist frequently with mental troubles. Syphilis, benign or virulent, may accompany mental diseases. The most frequent mental disorders are melancholia, mania, hallucinatory delirium and stupor. These psychoses terminate nearly always in recovery. Specific treatment, according to numerous clinicians, shortens the duration of the malady. Syphilis can determine mental disorders (melancholia, suicide) by hypochondrial ideas.

2. *Curve of Intellectual Work.*—The perpetual conflict between psychologists and philosophers is well known. The systematic researches of Kraepelin are of a nature to throw light on modern psychology. He has endeavored to find the laws governing the normal human mentality, and to this end has for some years, at Heidelberg, pursued investigations in the question of intellectual work.

I. *The curve of intellectual work.* Kraepelin attempts to show what changes are reproduced in intellectual work under the influence of internal causes without changing external conditions.

In order to measure intellectual work one is given simple problems, of which he is to solve the greater number in a given time, usually five minutes. The resulting curves show various oscillations, which he endeavors to explain. Nearly all the curves descend towards the end This is due to fatigue. Some do not descend. This is due to practice. In general, if exercise dominates the curve rises. It descends when fatigue is stronger than practice. Generally the curve descends in the evening more quickly than in the morning. But this differs in different individuals according as they are accustomed to working in the morning or in the evening. In general, we consider the lowering of the capacity for work as a symptom of fatigue only when it progresses with the work, and not when it decreases as the work is prolonged. The introduction of food also, independent of fatigue, diminishes the capacity for work. Digestion and the distribution of blood play an important rôle here. These conditions and others, such as sadness, distraction, resemble fatigue, but the curve rises as it does after poisons (ether, alcohol) when their effects have worn off. Fatigue can, too, be compensated by an effort of the will. One could say in general that the work curve was an expression of the relations between practice and fatigue, but this is not strictly true. Practice leaves durable traces in the mind, while fatigue is removed by sleep and food. Thus we arrive at the result that with more and more practice the curve begins to descend more and more quickly, due not to greater fatigue but to a lessened influence of practice. If work is prolonged the descent of the curve is more and more rapid. The relation between fatigue and exercise is more and more unfavorable. This effect could be produced within certain limits by the decrease of practice, but this decrease for advance degrees of practice is very slow, and does not explain the sudden descent of the curve. This would seem to be because, as the fatigue increases, the interior resistance to the work increases so that in order to accomplish the same work one must expend a greater and greater force. Nevertheless this seems improbable from the work of Oseretzkowsky and Kraepelin. Rivers and Kraepelin found that the loss of the effect due to practice for each half hour of work was the day after thrice as great for a work of two hours as for a work of a half hour. It seems that the capacity of practice and fatigue are the common expression of certain fundamental qualities of the psychic personality.

II. *The pauses in the curve of intellectual work.* The phenomena of fatigue disappear quickly, while practice leaves durable traces. The persistence of these traces may extend over a long time. After two exercises of adding of two hours each Kraepelin found the remains of practice three months later. Intervals affect the practice curve variously. The loss is greater after certain hours of the day than after a night's sleep. Habit also modifies the curve. By short pauses the effects of fatigue can be to an extent balanced. Intellectual work seems to produce general fatigue, while muscles may be recuperated by stopping their use. There is a *pause favorable* depending on the duration and nature of the work, and which determines the relation between practice and fatigue. Lindley found this pause in three subjects from fifteen to sixty minutes. Before the *pause favorable* the residues from practice and fatigue are in equilibrium. If the work is interrupted at this moment it should have the same value as taken after this pause—it is called the pause of equilibrium. The greater the fatigue the more the influence of rest is noticeable.

III. *Construction of the curve of intellectual work.* The construction of the curve is very difficult, the different factors causing variation are so closely connected. For instance, in studying the effects of pauses it is difficult to estimate their true value, because after a pause the other factors have not remained unchanged.

What follows is a brief discussion of the methods of obtaining a normal curve and curves of practice, habit, excitation, will and fatigue.

3. *Homicide by a Paralytic.*—The author describes a case of attempted homicide by a paretic, and cites several cases from the literature. He concludes that dangerous tendencies are not rare, and argues for more careful surveillance of this class of patients. W. A. WHITE.

Review of Neurology and Psychiatry

(Vol. 3, 1905, No. 2, February.)

1. The Endocellular Fibrillary Reticulum and its relation with the Fibrils of the Axis-cylinder. A. DONAGGIO.
2. Some Aspects of Alcoholism (Continued). A. HILL BUCHAN.
3. A Micro-chemical Examination of the Phosphorous in Blood-Clots. JOHN TURNER.

1. *The Endocellular Fibrillary Reticulum.*—The author believes it is quite evident that the fibrils of the axis-cylinder take their origin from the endo-cellular reticulum. The seat of origin varies in different cells, but is always demonstrable under his special methods of staining, which he describes in detail in his paper. His researches prove the existence of two types of cells: The first is characterized by cells provided only with an endo-cellular fibrillary reticulum, wihch is in relation with the fibrils of the protoplasmic processes; the second is more complex and includes the great majority of the nerve-cells. These possess two fibrillary systems, (*a*) fibrils forming the endo-cellular reticulum; (*b*) fibrils passing through the cell, but preserve their individuality. Donaggio states that if we admit that the fibrils possess the function of conducting nerve-currents, it is fair to infer that the endo-cellular fibrillary reticulum is an apparatus of reception and of synthesis of the stimuli transmitted to it by the cellulipetal paths.

2. *Some Aspects of Alcoholism* (Continued).—Continuing his statistical study of the cases of alcoholism which he began in the January number, 1905, Buchan states that out of the total number of 418, 62 had definite symptoms of neuritis. The average age of the cases of neuritis for both sexes was 40 years. As regards subjective sensations under the nervous system, numbness was found more frequently than pain. Sensibility to tactile impressions entered as normal in 19 cases, delayed in 3, and impaired in 9 cases of the 418. Hyperesthesia to deep pressure on muscle and nerves played rather a prominent rôle, and was found in all 62 cases. The tremor movements rendered investigation as to the occurrence of ataxia difficult. The knee-jerks he found increased in a large number of cases. There was a preponderance of affection of the lower over the upper extremities, and of paralysis of the extensors over the flexors, and also of the posterior over the anterior aspects of the limbs. The most frequent departure from the normal was the disturbance in regard to pain in the lower extremities, this being almost always in the form of muscular hyperesthesia. And the almost invariable seat of pain was in the calves. As to mental symptoms, his observations did not materially differ from the usual picture of Korsakoff's psychosis. Of the 62 cases 31 recovered, 13 improved, 7 were sent to an asylum, 3 transferred to a general ward and 8 died.

3.. *A Micro-Chemical Examination of the Phosphorus in Blood-Clots.*—Turner has made a series of experiments on blood-clots, trying to discriminate between the clotting which is the result of vital action and that which occurs on the application of the alcoholic fixative used in preparing the tissues for sections, or after the death of the cells. By means of Macallum's phenyl-hydrazin hydrochloride test the phosphorus-containing material in the coagulum is stained green. On examination of the ante-mortem clots the coagulation consists of a nucleo-proteid substance which stains green. In the post-mortem clot and that which occurs on the application of alcohol to the tissues the coagulum consists of a substance absolutely devoid of phosphorus, and therefore not nucleo-proteid. but which would seem to be the result of distintegration of the red corpuscles. "When it becomes a question as to the significance of the nucleo-proteid clots," the author concludes, "we still have no tests to allow us to differentiate between the coagulation in a moribund or recently dead subject, which may have no pathological significance, and that which may occur during active life, where it would have a very decided pathological significance. GLASCOCK (Washington).

Allgemeine Zeitschrift fuer Psychiatrie

(Vol. 62, 1904, Nos. 1 and 2.)

1. Chronic Paranoiacs in their Legal Relations, Civil and Criminal. L. W. WEBER.
2. Brain Weight and Insanity. RICHARD MITTENZWEIG.
3. On Color Hallucinations. GEORG LOMER.
4. What Arrangements in the Asylum do our Present Therapeutic Methods Require? WURTH.
5. Dementia Precox or Brain Tumor? OTTO KAISER.
6. Mental Condition of Deaf Mutes. H. KORNGELD.
7. Persons Ordered Committed to German Asylums for Examination as to their Sanity.
8. Simulation of Mental Diseases. ERNST BISCHOFF.
9. Simulation of Attacks of Pain in Morphine Habitué. NERLICH.
10. Endogenous Symptom-Complexes in Exogenous Disease Forms. FAUSER.

1. *Chronic Paranoiacs in their Legal Relations.*—Descriptions of the cases of two chonic paranoiacs, the one having a history extending over 25 years, the other over 20 years. The one, besides excellent ability in his proper trade, that of a mechanic engaged in sewing machine construction and repair, was able to earn considerable money as a system of irregular medical practice evolved by himself. From time to time he had periods of disturbance with hallucinations and illusions, which led him into making false accusations and entering into litigation which resulted in landing him in an asylum, from which he later was either discharged or escaped. Except for the influence of his peculiar ideas, his judgment remained good and he showed no serious mental failure. The second case is that of a man springing from a peasant family having strong religious opinions, who entered upon theological studies, but being dismissed from the seminary on account of insubordination and repeated disagreements with his superiors, launched out as an independent preacher. and, going about clad in sackcloth, disturbed the meetings of the regular congregations, whom he denounced without cessation. As he was continually in conflict, he was committed to an asylum, where he remained three years. After a short period of liberty he was committed to another asylum, from which after another three years' sojourn he escaped. He then wandered about for two years preaching, and gained many adherents, who supported him and gave him considerable sums of money, nearly all of which he spent in having his own peculiar polemics printed. He again became involved in conflicts with the church authorities, and was again committed. In spite of his peculiarities he possessed real ability in composing and preaching sermons, and had a remarkably retentive memory. Even after 20 years he shows no real mental failure, and has apparently

never had hallucinations or illusions. The author discusses these cases, neither of which he thinks can be considered as a querulant, but rather as an example of chronic paranoia, the one with, the other without hallucinations in their forensic relations, and concludes that while at times their conduct constituted a public nuisance, if not a danger, there is considerable question as to whether they should be held as entirely irresponsible, and whether for them life internment is necessary or desirable.

2. *Brain Weight and Insanity.*—In order, if possible, to answer the question whether brain weight below some definite figure, when found postmortem, justifies the conclusion that mental disease had been present during life the author compares the weights determined by Marchand for the normal brain, at diffferent ages in the two sexes, with those found for the brains in 1,123 cases of insanity—dementia paralytica 467 cases, dementia senilis 332 cases, organic psychoses 139 cases and functional psychoses 185 cases—examined at Herzberge from 1893 to 1901. His results are classified according to age, sex and character of mental disease, and arranged in tables. Marchand found in the normal individual the lowest brain weight 1,000 grms. for men and 950 grms. for women. Among the insane the author found brain weights of under 1,000 grms. in dementia paralytica in 2.1 per cent. of all cases, in dementia senilis in 1.6 per cent. of all cases, in organic psychoses under 60 years of age no case, over 60 years in 2.8 per cent. of the cases, in functional psychoses no weight below 1,000 grms. was found. He concludes that in males at any age brain weight below 1,000 grms. points to the probability of previous mental disease, probably dementia paralytica or dementia senilis, or if the individual is over 60 years of age possibly an organic psychosis. In a person known to have been insane, below 60 years of age a weight of 1,150 grms. or below, above 60 a weight of 1,100 grms., allows the exclusion in all probability of a functional psychosis. In the case of a woman, the age is first to be considered. Under 60 years, brain weight under 1,000 grms. points with much probability to preëxisting mental disease, probably dementia paralytica or organic psychosis. Over 60 years, a weight below 950 grms. points to previous psychosis, probably either dementia senilis or organic psychosis.

3. *Color Hallucinations.*—Color hallucinations are to be divided into those due to known and those from unknown causes. After reviewing the various theories as to their causation, especially considering the occurrence of such hallucinations in neuroses and psychoses, the author relates the case of a young woman of strong nervous heredity in whom he could find no hysterical stigmata, but who was committed to the asylum for a psychosis probably to be classed as dementia precox, but who after five years had not become demented. This patient, after a period of apparent lucidity, was seized during a menstrual period with an attack of excitement during which she insisted that she saw everything green in color. Regularly three times a day, about an hour before meals, she had a period of great excitement and confusion. These attacks recurred during fourteen days, when she became quiet. The green vision persisted for about four weeks, and during the next menstrual period there was a short attack of excitement less marked than the previous ones. Examinatoin of the eye-grounds showed nothing abnormal. The author ventures no definite explanation of the phenomenon, but is inclined to refer it to a toxic or vasomotor cause.

4. *Arrangements in the Asylum.*—The author thinks that separate asylums for acute and for chronic patients are impracticable, if not unwise. He discusses at length the proper arrangements for carrying out the newer methods of treatment by confinement in bed and prolonged baths, where possible in an observation ward, isolation and keeping in cells being dispensed with as far as possible and restraint being rele-

gated to the past. Of special interest are his suggestions for the management of disturbed and unclean patients. He estimates the proportion of patients in a mixed asylum who can with advantage be subjected to bed treatment as high as 60 per cent.

5. *Dementia Precox or Brain Tumor?*—Description of the interesting case of a young woman in whom for ten years typical symptoms of dementia precox, with eventually an extreme grade of dementia, were present. She then began to have Jacksonian convulsions specially affecting the right side of the face and right arm, which continued at intervals until her death about eleven months later. The eye-grounds were normal. Postmortem examination showed a diffuse glioma with hemorrhage and softening affecting nearly the whole of the right hemisphere. The occurrence of the Jacksonian attacks on the right side, and not on the left, as would be expected from the location of the tumor, the author thinks due either to pressure or to an existing meningitis, as otherwise no macroscopic changes were evident on the left side. A microscopical examination does not seem to have been made. He states, however, that neither pyramidal tract appeared quite normal, and that he was able to assure himself that decussation was present. Discussing the question as to whether the tumor was actual cause of the initial mental symptoms or only a complication of preëxisting dementia precox, he inclines to the opinion that since the neoplasm was of probably very slow growth, the former view of the case is the correct one.

6. *Mental Condition of Deaf Mutes.*—Continued article.

7. *Persons Ordered Committed to German Asylums.*—Statistical table.

8. *Simulation of Mental Diseases.*—Continued article.

9. *Simulation of Attacks of Pain in a Morphine Habitué.*—An account of the case of a man of considerable education who had led the life of a swindler, had been frequently in prison and had contracted the morphine habit, who, interned at Waldheim both for cure of the drug addiction and for an opinion as to his mental condition, came under the care of the author. After careful study of the case the conclusion was reached that he was not insane, but an ordinary liar and swindler, and that his pains were simulated to secure morphine, to arouse sympathy and to escape or lessen punishment. The author does not, however, deny that the patient may belong to the class of psychically inferior, and thinks that institutions for the care of such people would afford great relief to doctors and to the public generally.

10. *Endogenous Symptom-Complexes in Exogenous Disease Forms.*—Description of a case of general paresis which began suddenly and for a year presented symptoms of manic depressive insanity without any of the somatic signs of general paresis, and with no impairment of memory or intelligence. The patient was sent home apparently convalescent. Shortly afterward he had a convulsive attack followed by typical symptoms, both somatic and psychical, of dementia paralytica. The author takes occasion to discuss the psychological mechanism of the production of various morbid mental manifestations in the so-called endogenous forms or degenerative insanities. He finds theoretically nothing opposed to the practically well-known fact that symptoms supposedly characteristic of these endogenous forms often occur both in general paresis and in other insanities due to exogenous causes. ALLEN (Trenton).

Miscellany

MENTAL DISTURBANCES IN REVIVAL AFTER HANGING. B. Bayerl (Wiener klinische Rundschau. Volume XIX, No. 4).

A young man, upon the restoration of the vital functions after an attempt to commit suicide by hanging, displayed a maniacal condition, with dilated, non-reacting pupils. After a number of days the mania subsided, and he had never showed any mental disturbance before or afterward. His arrest on a charge of murder was the occasion of his deed.

JELLIFFE.

PATHOLOGICAL SLEEP IN CASES OF CEREBRAL TUMOR. Fr. Franceschi (Riv. di pat. nerv. e ment., October, 1904).

Franceschi describes a case of cerebral tumor attended from the first by a tendency to sleepiness, and raises numerous questions with regard to the nature both of physiological and of pathological sleep. He notes that while all authors mention pathological sleep as a symptom of cerebral tumor, it is usual to note its existence without any investigation of its cause or any explanation of the different stages of the disease at which the symptom shows itself. Some authors do not distinguish clearly between somnolence, sleep and coma, and it is, therefore, impossible to draw exact conclusions from cases reported by them. The principal questions raised are those of the mechanism of sleep and the effect of the localization and nature of cerebral neoplasms in producing sleep. Righetti has shown that pathological sleep is a more common symptom of tumors in the third ventricle and the hypophysis than of those situated elsewhere. In this case the patient suffered from somnolence, and later from mental torpor, for five months before an affection of vision and the increasing severity of the other symptoms led to her admission to hospital. At that time she presented the appearance of a continuous and tranquil sleep. She was capable of being aroused, but quickly slept again. Later she suffered from headache and vomiting, and she died of bronchopneumonia a month after admission. She was conscious when awake up to the end of life and frequently executed voluntary movements. Her sleep was much disturbed by the bronchopneumonia. It was, therefore, clearly sleep and not coma. At the autopsy a sarcoma was found originating probably in the dura mater, and afterwards involving the base of the third ventricle and the neighboring parts. The tumor was of such a size and in such a position as to be closely surrounded by the circle of Willis. The internal carotids were half their normal size, and the posterior communicating arteries were a quarter or a fifth of their normal size. The basilar arteries were compressed by the tumor. The most commonly-received explanation of sleep caused by a cerebral tumor is that it is due to increased intracranial pressure, but in this case there was no appearance at the autopsy of anything beyond slight increase of tension, and no likelihood that there was any appreciable increase of tension when somnolence was first observed. One of the best-established facts with regard to sleep is that it is attended, either as cause or effect, by anemia of the cerebral cortex. That theory of sleep which regards it as caused by anemia of the cortex fits best with the conditions observed in this case, and the frequency with which pathological sleep is associated with tumors of the optic thalami, the hypophysis and the floor of the third ventricle may be reasonably attributed to pressure on the circle of Willis, interfering with the circulation through the brain. Other theories—chemical, histological and evolutionary—of sleep causation the author accepts as complementary to and not incompatible with this. A much larger number of careful and accurately recorded observations is needed before each hypothesis can be credited with its true worth. Franceschi briefly reviews the opinions of those who maintain the existence of a special center for sleep located in the central grey substance of the brain, and points out that observations made in this case might well be cited in support of such a theory. JELLIFFE.

THE CURABILITY OF EARLY PARESIS. C. L. Dana (Journal A. M. A., May 6).

The author suggests that paresis, like tabes—with which it is closely related as a parasyphilitic disorder—may be arrested in its earlier stages.

By "arrested" he does not mean the well-known remissions of the disease; in these, he says, there still remains a certain amount of paretic mental impairment, but he rather means a complete disappearance of all evidence of degenerative changes in the brain. He reports a number of cases illustrating his contention, in which symptoms decidedly indicative of paresis appeared, characteristic mental changes, convulsions, Argyll-Robertson pupil, etc., but which disappeared under treatment, and the patients remained well for various periods under observation. The treatment generally consisted in complete change of life, antisyphilitic medication, preferably hypodermic, hydrotherapy and attention to general nutrition. He says there is no *a priori* reason why paresis in its early stages may not be sometimes cured, and he holds that the cases he here reports point that way and indicate the importance of an early diagnosis and treatment of this disorder which has been heretofore considered incurable.

THE CASE OF JOHN KINSEL. G. B. Cutton (The Psychological Review, September, 1903).

The history and discussion of a very interesting case of "double personality." Subject, 32 years old. Born of respectable and well-to-do farming people of New England. Alcoholism and insanity, to marked extent, in paternal and maternal ancestry. During early childhood several convulsive seizures. Headache, vivid dreams, stuttering, somnambulism and general nervousness. Cataracts, both eyes. Prepared for and entered college at twenty. Fair scholar. "Sleeping state" first noted by fellow-students during freshman year, increasing in frequency and degree till one year after graduation. In this condition there was marked improvement of memory and brighter intellectuality. Oftentimes it became necessary for him to attend examinations in hypnotized state, having heard the necessary lectures while in "sleeping condition;" thus normally, with very limited knowledge of subjects for tests. Ordinarily prudent as to expenditures, gentlemanly and of good habits; in secondary state he became boisterous, coarse, extravagant and given to drink. Condition of eyes stated to have been important factor in auto-hypnotism. Attacks infrequent, while at work farming, during vacations. Lessening of mental strain, etc., thought to be the reason. During junior year, several epileptic seizures. Following graduation and while a divinity student, several attacks of abnormality. Later, dypsomania; treatment by hypnosis. At present subject successfully engaged in teaching, apparently cured and well in every respect except eyes, which are much improved, as result of operative procedure, etc. J. E. CLARK (New York).

THE FEAR OF DEATH. J. Leonard Corning (Journal A. M. A., May 6).

Corning discusses the morbid exaggeration of the fear of death, which he considers due to a neuropathic basis inherited or acquired. In animals the fear of death is dependent on its imminence; in man it is sometimes a permanent obsession, but it is even then usually absent in the actual process of dying, the dulling of consciousness at that time and other dominating physical conditions accounting for this fact. He reports a case illustrating what he considers the essential psychology of the morbid dread of death, in this case even exciting suicidal impulses—death to escape death. In treating this condition he would suggest the thought that sleep is a sort of death, and unconsciousness, whether lasting or not, a boon. His treatment was to prevent sleep until it was sought imperatively, and was based on the theory of proving experimentally that the temporary unconsciousness of sleep is the remedy for curable shortcomings and convincing the reason that the more lasting unconsciousness of death is only the supreme antidote of the irremediable breakdown of the organism, and therefore supremely benevolent in its essential nature.

Diagnosis of Death by Drowning. Revenstorff (Münchener medicinische Wochenschrift, March 21, 1905).

Revenstorff bases his conclusions on the fact that hemolysis of the blood is one of the earliest macroscopical signs of putrefaction, being due not to physical influences or to autolytic action, but to bacterial action. It appears in the blood of the various vessels in the following order: Portal vein, right heart and afferent vessels, left heart and arterial system. What the author terms the "drowning hemolysis" occurs by a foreign fluid getting into the capillaries or lung tissue, thus coming in contact with the blood and, if the difference of concentration between the foreign fluid and that of the body is sufficiently great, dissolving the blood corpuscles involved. This hemolysis, contrary to the cadaver hemolysis, appears in higher degree in the serum of the left heart. Lung tissue juice, pleural transudate and pericardial fluid are free from admixture with the drowning fluid if they do not show any free hemoglobin. Edema caused by the presence of water always has a serum containing hemoglobin, the true lung edema having a serum without color. Consequently, if no pigment shows in the contents of the portal vein, but drowning hemolysis is evident in the blood of the heart, the proof is positive that the blood has ceased to move in the heart before the drowning medium was diffused. The medico-legal applications of this conclusion are obvious. Jelliffe.

Pneumonic Paralysis. Fedeli (Rif. Med., Feb. 25, 1905).

The author reports a case of right hemiplegia occurring in the course of pneumonia in the case of a man aged 65, and due (as proved by necropsy) to hemorrhage in the left internal capsule. He then discusses the subject of pneumonic paralysis generally. A short review of various cases teaches that there are several varieties of paralysis occurring in pneumonia. Those occurring at the beginning or in the course of the disease have a different symptom-complex and a different prognosis from those which happen during convalescence. The latter are diffused in type, with a tendency to become fixed in the lower extremities, and almost always recover. The former are more often monoplegic or hemiplegic in type and may end in death. Probably the paralyses of convalescence are of the nature of a neuritis or polyneuritis, which may or may not be of the ascending type, and occasionally leading to poliomyelitis. The muscular atrophy which sometimes accompanies these forms of paralysis tells in the same direction. As to the more serious group of paralyses—those occurring in the beginning or during the course of pneumonia—many of them are due to cerebral hemorrhage, others to thrombosis, embolism and softening. Of those which recover some may be explained by localized anemia of reflex origin (for example, in children), or due to vascular changes (in old people). Even the toxemic paralyses probably act chiefly in virtue of some previous vascular weakness forming a locus minoris resistentiæ. Hysteria is not a very probably cause, and may be largely discounted in these cases and it seems fairly certain that whatever be the real pathogenesis of pneumonia, it must in their case be something different from that of old people. Jelliffe.

New Discoveries and Advances in the Field of the Pathology of Rabies. E. Bertarelli (Wiener klinische Rundschau, Volume XIX, No. 4).

This article is a survey of the researches in the pathology of rabies made for the past two or three years in Italy. It would seem that they are unanimous in declaring that the Negri bodies are never found in animals free from rabies, and that if they are not found in those affected it is always in cases where the search has not been an exhaustive one. The hippocampus major is their chosen site, and examination of it would be fairly conclusive, entirely so if the finding proved affirmative, but if negative a supplementary test by inoculation of a rabbit would be advisable, as well as an examination of the cerebellum and ganglia. Fragments of the hippocampus may be hardened in saturated sublimate solution or in absolute alcohol and stained by the Mann eosin-blue method, or by fixing the fragments in osmic acid, leaving them for a few hours in alcohol. Sections can then be made by hand and tested with glycerin. The evidence is that these bodies cannot be produced artificially, and that they never occur except in rabies. Volpino's experiments indicate that each of them contains a central basophile small body, the two parts of which have different staining affinities. The saliva of patients with rabies is sometimes, but not always, virulent. Some months ago Nitsch undertook to discover the truth as to the harmlessness of fixed virus by injecting it subcutaneously into himself. At the time of writing no effects of any sort had appeared. Valenti has been able to neutralize the virus of rabies in a test tube with quinine, and in the living body as well, but no other alkaloid has had the same effect. JELLIFFE.

PNEUMATOCELE OF THE CRANIUM. L. L. McArthur (Journal A. M. A., May 6).

The author reports an operation for this condition, and discusses the diagnosis and treatment. The etiologic factors are chiefly two—traumatism and sudden increased pressure within the buccal and oral cavities. Fifty per cent. of the reported cases have occurred spontaneously, so far as history of injury or inflammatory conditions are concerned. During the growth of the tumor sneezing or blowing the nose may cause an appreciable increase of the size of the tumor, or external pressure may cause escape of air into the oral cavity. Because of the separation of the periosteum from the bone osteophytes may be produced, and this accounts for the irregularities felt when the tumor is collapsed. With modern antiseptic methods the treatment has become more simple and successful. In nearly every case a perfect cure can be effected if a free opening is made with due care so to dispose the incision as to make the point of final healing immediately opposite the bony perforation through which the air entered the tumor. McArthur quotes the conclusions of Costes, of Bordeaux, as follows: I. Pneumatoceles are very rare. 2. They always depend on perforations of the bony walls. 3. They are always tympanitic. 4. They are more or less reducible by pressure. 5. They can take their origin only from the mastoid or the frontal sinuses. 6. They are of very slow and indolent formation. 7. They are never dangerous except from complications (infections). A bibliography is appended.

MYASTHENIA GRAVIS WITH SPECIAL REFERENCE TO THE OCULAR SYMPTOMS, AND A REPORT OF A CASE INVOLVING THE EYES ONLY. Mortimer Frank (The American Journal of the Medical Sciences, April, 1905).

After a critical review of the literature the author reports a case presenting this condition in a girl 10 years of age. At present she has double ptosis, most marked on the right side, paresis of all the extrinsic muscles of both eyes, and a marked divergent squint of the right eye. There is complete preservation of the functions of the intrinsic muscles of the eye. The first symptom, the ptosis, began when the patient was 6 years old, and would at first intermit for a day or two at a time. From the beginning there was no evidence of ptosis on arising in the morning, and after resting her eyes the movement of the lids appears to be normal, but they are rapidly exhausted. There was also observed some weakness in the orbicularis palpabræ and occipito-frontalis muscles. Other bodily functions are normal, but a feeble myasthenic reaction has been demonstrated at times. C. D. CAMP (Philadelphia).

MYASTHENIA GRAVIS WITH PARALYSIS CONFINED TO THE OCULAR MUSCLES. William G. Spiller and E. U. Buckman (The American Journal of Medical Sciences, April, 1905).

A groceryman, aged 33 years, first noticed April 26, 1904, a dimness and blurring of vision. He had for some time previously had severe headaches, not associated with any ocular symptoms. In November, 1905, there first developed a ptosis of the left eye, and occasionally in the right eye. Examination at this time revealed almost complete paralysis of the inferior rectus muscles of the right eye, but the ocular palsy has seldom been the same at any two examinations; sometimes one muscle is paralyzed, then another. When he takes off his glasses the upper lid of one eye. depending upon which eye he has been using last, begins to fall and gradually there is complete ptosis; while this is occurring the upper lid of the other eye gradually droops until ptosis may be complete or nearly complete on this side also. After the upper lids have fallen, if he closes his eyes a few minutes he can again open them fully. There is no history of syphilis or alcoholism in the case, and large doses of iodid have been given without result. Examination of other parts of the body is negative, except that there is some exhaustion of the left sternocleidomastoid muscle by the Faradic current. CAMP (Philadelphia).

SIMULTANEOUS OCCURRENCE OF SYPHILIS AND TABES. C. Adrian (Zeitschrift f. klinische Medicin. LV, Naunyn Festschrift).

Adrian tabulates the particulars of 96 cases of tabes. In 16 there were active manifestations of syphilis in the cerebrospinal system, in 15 there were active manifestations elsewhere, all these being established anatomically, and in 65 there had been evidence of active constitutional syphilis accompanying tabes. In more than 12 per cent. the syphilitic infection had been ignored during life. The author concludes, among other things, that these manifestations of florid syphilis indicate an earlier, and therefore more curable, stage of tabes. JELLIFFE.

SYPHILITIC EPILEPSY. J. T. Moore (Journal A. M. A., June 10).

This is an account of a case of epilepsy occurring in a man 35 years of age who had suffered from an alleged fracture of the right frontal bone about five and a half years before, the epilepsy dating back about three years. Syphilis was denied. The convulsions began on the right side of the face and extended from there over the rest of the body. The focal symptoms did not warrant operation, and as there was some roughening of the tibia and some enlarged glands, he was given iodid of potash in gradually increasing doses. The attacks, however, became more frequent and severe, in spite of the use of bromids, chloral, etc., and in a few days he was put on 1-10 grain doses of bichlorid of mercury, with the result of rapid improvement and complete cessation of the attacks. Syphilis was suspected in this case because of the age at onset, the other symptoms strengthening the suspicion, which was fully confirmed by the results of treatment.

BRAIN HEMMORRHAGE. W. A. Dickey (Journal A. M. A., May 13).

Dickey considers alcohol and syphilis the two most prominent causes of the arterial degeneration favoring brain hemorrhage; next to these comes chronic interstitial nephritis, and after these a multiplicity of other factors leading to arterial decay. Still another factor is required in all cases, namely, increased intracranial blood pressure, such as may be caused by muscular effort, indigestion, etc. Beside the prophylactic measures, such as quiet, avoidance of whatever may cause cerebral congestion, increased work of the heart, etc., he advises for the attack itself the use of powerful cardiac depressants, naming, in the order of their importance, aconite in full doses, veratrum viride, gelsemium and venesection. Gelatin is, he thinks, too slow in its action and not always practicable. Cathartics should be avoided for the first few days, and he sees little utility in an icecap to the head. The patient should be kept absolutely quiet in bed for ten days or two weeks.

CEREBROSPINAL FEVER. J. C. Wilson (Journal A. M. A., April 29).

The author reviews the history, causes, symptoms, treatment, etc., of epidemic cerebrospinal meningitis, a disease which has at present a special interest on account of the lately occurring epidemic in New England and New York. While it has been recognized for about a century, most of our knowledge of the disorder has been acquired of late years, and largely through the work of our countrymen, Councilman, Mallory and Wright. Councilman's recent paper (Journal A. M. A., April 1, 1905) is referred to by Wilson. Formerly the communicability of cerebrospinal meningitis was doubted, but it is now admitted that if the nose, ears or lungs are affected it may readily be conveyed from one person to another. Second attacks are very rare; it is probable that one attack confers a persistent immunity. The germs are found only in connection with the lesions of the disease, but mixed infections are not uncommon. The symptoms are most diverse, there are no prodromes and the period of inoculation is unknown. In the malignant cases the symptoms of inflammatory lesions of the brain and cord and those of a general malignant infection are both overwhelming. The author goes at some length into the description of the general symptoms and those of the anomalous types, the fulminant form, the abortive, the intermittent and the chronic types. Few diseases vary more in their severity and mortality, or are followed by more complications. The diagnosis may be difficult, but if meningitis be present it is not usually embarrassing during an epidemic. In doubtful cases lumbar puncture should be resorted to, and the presence of the meningococcus in the cerebrospinal fluid ascertained. The differential diagnosis between this form and tuberculous meningitis may in some cases be far from easy without this. The mortality of different epidemics varies between 20 and 75 per cent.; the average is estimated by Wilson as near 40 per cent. In the mildest cases no treatment is required; in the malignant ones none is effective. Quiet, nutritious diet, cold applications to the head and spine, laxative doses of calomel in the beginning of the attack, and opium are recommended, the last-named drug being regarded as indispensable. For prophylaxis, cleanliness and avoidance of overcrowding in times of epidemics, isolation and sterilization measures, and in case of successive attacks in the same family, abandonment of the dwelling and thorough disinfection are advised.

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Original Articles

A CASE OF CEREBRAL TUMOR PRESENTING CONFUSING SYMPTOMS.[1]

BY FRANK R. FRY, A.M., M.D.,

OF ST. LOUIS.

PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM, MEDICAL DEPARTMENT, WASHINGTON UNIVERSITY.

Synopsis—Tumor symptoms were evident. The gait and other symptoms led to its localization in the left side of the cerebellum. An exploration failed to discover it. On autopsy the neoplasm, 5 cm. x 6 cm. in size, was found to occupy the base of the third frontal convolution on the left side, growing especially in the operculum and triangular lobule and compressing the ascending frontal and superior temporal convolutions and the island of Reil. There were no sensory, motor or speech symptoms to draw attention to this locality. The subject was left-handed.

I first examined the patient with Dr. U. S. Smith, June 11, 1903. Dr. Smith had made a diagnosis of brain tumor, leaving the question of localization open. The tumor symptoms were decided: headache, vomiting and vertigo, recurring in typical exacerbations. I noted at this time several additional features, as follows: the patient's head frequently assumed a certain twisted position with the occiput turned to the left shoulder and the chin slightly elevated and turned to the right. He had a staggering gait and in walking invariably veered to the left side and frequently had a tendency to stumble and fall to that side. The knee-jerks were very low and no sensory changes could be found.

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[1] Read at the meeting of the American Neurological Association, June 1, 2 and 3, 1905.

The history suggested that tumor symptoms had begun two years or more previously and had been becoming rapidly more urgent in the last few months.

He was placed in St. John's Hospital. After several weeks of observation I still figured the localization in the left cerebellum. (An intracerebellar tumor.) Dr. Paul Y. Tupper operated accordingly, making a thorough exploration, and found no tumor. The patient was for a time much relieved from pressure symptoms. During this time I recanvassed the case, and decided that the other side of the cerebellum should be examined if the opportunity to do so were afforded. There were, however, various delays. Meantime the patient became worse and died about five weeks after the operation with cerebral edema. An autopsy was made. Dr. Carl Fisch furnished the following report on the specimen:

"The tumor occupies the greater part of the site of the left inferior frontal convolution. It is of an orange shape, the larger diameter being about 6 cm., the smaller one, 5 cm. It lies in the plane of the pars triangularis and the pars opercularis, abutting with part of its periphery on the ventral end of the central fissure. The surface toward the Sylvian fissure as well as the dorsal surface are covered with a thin layer of tissue of pial origin. On the dorsal surface there is present in one place an adhesion to the dura. On the rest of its surface the tumor is sheathed with a portion of the remaining tissue of the third convolution. The thickness of the brain tissue between the tumor and the sulci varies from 1 to .05 cm. The pars orbitalis is also encroached upon, although not to the same extent as the other portions of this gyrus. The growth has compressed a portion of the precentral gyrus, and also the superior and middle temporal, and the convolutions of the island of Reil. The tumor is everywhere sharply outlined against the surrounding tissue in contact with it. There is, however, no macroscopically visible capsule. Its consistency is very soft and shows on section a granular appearance of grayish color (in the formal-hardened specimen) with small numerous reddish dots and streaks. The section, too, does not show any distinct capsule, and where there is continuity with the brain tissue the tumor tissue is sharply defined from the latter.

"The microscopic examination suggested at first glance a carcinomatous formation. There are found clusters of large cells with ill-defined outlines, that lie closely packed together in irregular smaller and larger masses. They have a well-staining granular protoplasm and a large vesicular nucleus. These masses of cells are imbedded in a stroma of myxoid character with small blood and lymph vessels permeating it. Closer inspection revealed that the large epithelium like cells could be followed up into gradually thinning threads and strands, that anastomosed with each other and showed the character of the cells changed more or less to a spindle shape. In other cases all of the cells approached the spindle type. It was found, too, that they were very often grouped concentrically around small bloods vessels and around lymph vessels. They were found also isolated in the stroma adhering to fine fibrous elements, a finding that was especially observed at the anterior periphery of the tumor. There a fine delicate form of membrane was found, of the structure of the pia mater, from which many of the large cells were formed and even arranged in groups. Similar appearances were also obtained in those portions growing into the brain tissue. The brain tissue had given way by pressure-atrophy, now and then small remnants of it remaining between the tumor cells. There was nowhere any inflammatory reaction around the growing tumor tissue. Perfectly normal ganglion cells were found in close contact with it. From these findings the tumor must be diagnosticated as an endothelioma."

After the autopsy I wrote a careful inquiry to the patient's brother and obtained a response which clearly revealed that our subject had been left-handed. To briefly quote my informant, he says: "He was very left-handed. He wrote with his left hand. He was left-handed altogether." It is very humiliating to confess an oversight of this importance, but probably worth while to mention it with the other instructive features of the case. The examination presented difficulties which will appear in what follows.

Physical examination: The pupils were always equal and responded to accommodation and to light usually rather sluggishly. The tendon reflexes were variable. The knee-jerks, at times quite easily obtained, were at other times only made evident by reenforcement. An ankle-jerk could often be obtained on each side; also wrist-jerk and elbow-jerk. None of them were ever lively, and I never could be positive that any of them were stronger on the right side. Even the supraorbital reflex did not seem stronger on this side. Inasmuch as I was always of the opinion that there was a slight right hemiplegia I examined repeatedly and carefully.

The lower portion of the face at times seemed quite paretic, the patient drooling constantly and holding a handkerchief to his mouth, always, by the way, in his right hand. On his good days the contrast was marked, there being then so little evidence of paralysis. At times I noticed a peculiar blank, expressionless condition of the right side of the face of brief duration.

The grip of the right hand seemed less than that of the left, but the difference if any was slight and difficult to determine on account of the listlessness of the subject. The patient seemed to use the right hand as much if not more than the left. As just stated he almost always carried his handkerchief to his mouth with his right hand and on some days the handkerchief was in constant requisition so that he used the hand a great deal in this manner. He usually reached for a glass of water with the right hand but supported it to his mouth with the left, holding it with the two hands as he drank. He was clumsy and uncertain in the use of both upper extremities. Although weak he had no tremor. There was at times a suggestion of a jerkiness, the same imprecision in movement that I have noticed in cerebellar cases; a dysmetria as it has been called.

His gait was not hemiplegic nor spastic, but at times he evidently shuffled the right foot more than the left in walking. He invariably gave way in the left leg in stumbling and falling. This happened so often and was so closely observed that there could be no question about it. The attendants and all others who saw him noticed the manner in which he drifted off to the left in walking. This tendency was so pronounced that at times he was unable to resist it except when some one constantly directed his attention to it. In rising from a chair he often lunged to the left and sometimes fell to the floor. His station was remarkably good; only a slight wabbling, and this was not increased, or at least but slightly, by closing the eyes.

The patient was rapidly getting weaker when he came to us. The attacks of vomiting, vertigo, headache and stupor were coming more frequently. Sometimes they were of a few hours' duration. At other times they lasted several days. He usually made a prompt rally from them. The contrast between his good and bad days was quite remarkable. On these better days he had no vertigo. At least he did not act as though he were dizzy and insisted that he was not, whereas on the bad days he frequently complained of it and exhibited the signs of it, i.e., a sudden swaying, tottery, limp state with momentary confusion and nausea which were readily recognized.

The twisted position of the head noted at our first examination was often remarked. Only a short time before the patient came under my observation I had read an article on the position of the head in cerebellar tumors in which the phenomenon was described as the "screwing out" or "screwing in" symptom according to whether the lesion were on the right or on the left side. Ours was invariably the "screwing in" position, suggesting a left-sided lesion. It was sometimes very pronounced, sometimes less so. I never could discover any resistance or spasmodic tendency in the muscles concerned in the movement. Speaking of this symptom Dr. T. Grainger Stewart says: "Though very characteristic, this attitude is neither constant nor pathognomonic. Occasionally in cerebellar growths the head is held in the reverse position and this 'cerebellar attitude' of the head is met with in cases of pontine, mid-brain, and more rarely, of fore-brain neoplasms. It cannot, therefore, of itself be used as a positive sign of cerebellar disease or as a definite aid in localization."

He complained of no tinnitus or deafness, and there was no difference in hearing between the two sides.

Dr. Meyer Wiener furnished the following ophthalmological report: "A subsiding optic neuritis on both sides. Right disc dark in color on nasal side and outline indistinct. Temporal half is atrophic, almost snow-white, swelling almost imperceptible. Left disc swollen about two diopters and still in active stage, but subsiding, outline indistinct, color brownish-red, no atrophy as yet, four small hemorrhagic spots radiating out from disc; no white spots."

There was no nystagmus. I often examined for it. No objective sensory phenomena were present except that he always complaind when the supraorbital nerves were pressed upon, and this apparent tenderness was the same on both sides. Believing that I had to deal with a cerebellar tumor it will be understood that I investigated the sensory condition of the face with proper care. It always seemed normal and I never found any difference between the two sides. Nor could I find any difference in the sensibility of the opposite side of other portions of the body.

The patient, aet. 43, was degenerate-looking, with stigmata about the head, face, hands and feet. The left occipital boss was much more prominent than the right, but there was no tenderness nor other sign to lead to the conclusion that this was other than one of several very evident asymmetries in his skull.

His mentality was a study in itself. His brother, an intelligent person, admitted that the patient was intellectually "not bright," but that he had only attended school about one year, and that he had lived his whole life in a remote rural region, and that at least some of his evident defectiveness should be charged to these meager opportunities. He had always been of an amiable and industrious disposition and quite a capable workman on a farm. He impressed us as being a very simple-minded, unsophisticated, stupid countryman. When feeling better, i.e., free from pain and weakness, he was fairly communicative and always good-natured. He was often somnolent and stupid, and it was then so difficult to hold his attention to serious matters that little could be accomplished in way of examinations. At other times, however, "Tom," as he was familiarly called by his hospital associates, became fairly alert, moving about almost to the extent of restlessness and manifesting a backwoods and childish curiosity in his surroundings, which coupled with his droll observations, peculiar drawl and clumsy antics made him the subject of much attention.

There were no essential speech defects. I conversed with him frequently, and in attempting to preserve a record of his mental condition I at times made written notes. For example, one morning a few days before the operation when I called he was expecting his brother "Bill" and becoming very impatient because he had not already reached the hospital. I suggested that Bill had not come to the city, but perhaps would in a few days. Tom retorted that he knew he had come because he had promised positively to do so on that particular day, and that he had not come out to the hospital but was "just foolen 'round down town in them stores." Tom then insisted that I should telephone to "the stores" and tell Bill to "come right out." When we asked what stores he replied "all of them, you just keep phonen 'round tell you strike him, then you tell him to come right on out here." He knew that his brother had business down town, but Tom was so "green" that he had no idea of the size of the city and of the complications of its telephone system. It was a fact that Tom's brother had promised for that particular day, and Tom had been carryin it upon his mind, attempting from time to time to aid his memory by asking others "What day is this?" "What day did Bill say he would come?" etc. His childish impatience was due to the fact that he was homesick and earnestly expecting Bill to take him home. On a former visit Bill had gone home without him, and Tom's mind was now bent on getting possession of Bill as soon as possible to persuade him to take him home. All of his mental processes were slow and his speech correspondingly so, but beyond this not defective.

Recapitulation: The tumor symptoms were evident. The localizing symptoms were a typical cerebellar gait and a constant tendency to deviate and to fall to the right in walking. The "cerebellar attitude" of the head was so pronounced that, coupled with the above symptoms, I gave it some localizing value. I also believed that there was present a homolateral atonia, although the tests by which I sought to establish this fact were not very satisfactory. The question was whether the contrast between the two sides of the body was due to a subtonic state of the left (i.e., the homolateral) side, or whether there was a plus-tonic state of the right (contralateral) side, or whether both of these conditions existed. As already intimated, I readily satisfied myself that the slight but evident weakness of the lower face was not due to the involvement of the 7th nerve. I therefore believed it to be due to pressure well forward upon the pyramidal fibers by the tumor, or possibly to internal hydrocephalus. In keeping with this view, I thought a slight hypertonia of the right side probable, although I found no increased tendon-jerks or other evidence of the fact, excepting the very suggestive manner in which the patient scraped the floor with his right foot in walking. The reflexes had all the variableness of a cerebellar tumor and this fact not only influenced my localization theory, but also led me to give them less weight in deciding the muscular state of the right side. The fact that he used the right or supposed paretic hand more than the left, I believed to be due to the presence on the left, in a decided degree, of the cerebellar symptom of atonia, or cerebellar paresis as it is called. The giving way of the left leg in walking exactly confirmed this theory. I had also noticed that in moving himself in bed the patient used the right hand more than the left members. I hence took for granted his right-handedness so decidedly that the thought of an inquiry about it did not enter my mind.

How were the symptoms present in this case produced? Was it an instance of so-called "frontal" ataxia, or possibly a kind of vertigo that is often difficult to distinguish from cerebellar ataxia, or were the symptoms after all cerebellar in origin notwithstanding no structural changes were found there.

In the winter number of Brain, 1904, Dr. James Collier, speaking of the false localizing signs of intracranial tumors, says: "In many cases of intracranial tumors of long duration, it is found post-mortem, that the posterior and inferior parts of the cerebellum have become pushed down and backwards into the foramen magnum, and the medulla itself being somewhat caudally displaced, the two structures together forming a cone-shaped plug tightly filling up the foramen magnum."

I believe this condition obtained in my case, although I did not notice the indentation by the edge of the foramen against the cerebellum which has been described in some cases. I am led to this opinion because in my close study of the case I was impressed that the symptoms were convincingly cerebellar.

MYSOPHOBIA, WITH REPORT OF CASE.[1]

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The recent achievements wrought in the field of psychiatry easily take precedence of all other departments of medicine in point of scientific interest and value. Until within quite a recent period the study of mental diseases appealed only to those members of our profession whose tastes and opportunities led them to select this form of practice. A radical change, however, is now taking place in the form of a general enthusiastic scientific awakening or spirit of inquiry relative to the nature and relationship of the various insanities, which is not confined to any special class of physicians, but permeates the ranks of the medical profession in all parts of the world. As a result, there never was a time when so much good work was being done as now; by not only the medical officers of our State and private insane hospitals, but also by the ablest alienists, neurologists, bacteriologists, clinicians and other medical investigators, whose combined efforts are now engaged in unravelling the secrets associated with mental diseases. The tendency of modern medical progress, therefore, is such as to bring psychiatry into closer relationship with general medical practice, by furnishing the general practitioner with more accurate and reliable knowledge concerning the clinical aspects of insanity and its allied neuroses.

The chief practical clinical results of this marked psychiatrical activity has furnished us the means of making finer discriminations in classification and diagnosis of the various insanities, as well as suggested methods for their more successful treatment. Moreover, it has also emphasized the important fact that while insanity is primarily a psychosis of the highest order, yet there are a number of morbid mental states which, while showing a marked kinship to insanity, are not usually regarded as such.

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[1] Read before the American Psychological Association, San Antonio, Texas, April 19, 1905.

These non-insane psychoses, however, as a rule, first come under the observation of the family physician, who, unfortunately, often fails to recognize their true psychological significance until the obsession-impulse, or fixed idea, which accompanies them, becomes so very conspicuous and persistent as to render the patient at times exceedingly troublesome, and even unsafe to be at large. It is, therefore, both gratifying and complimentary to our profession to note the change which is rapidly taking place in our conception of the term insanity, as well as our recognition of the close relation which exists between the psychoses and neuroses or those morbid mental states which formerly were not considered as belonging within the domain of the science of psychiatry.

Those of us, however, whose professional duties bring us in daily contact with persons suffering from the various nervous disorders cannot fail to recognize the confusion which exists in general medical practice concerning the diagnosis of such conditions as neurasthenia, hysteria and the different forms of insanity.

The extreme relational importance of these neurotic states with insanity, while being duly accepted by us, are not sufficiently recognized by the general medical profession as their significance justly warrants, hence with a view of emphasizing the close kinship that exists between them and other practical features, I am led to report the following case of mysophobia:

Miss A———, aged 25 years, single, brunette, occupation stenographer, was referred to me May 17, 1902, by her family physician.

Family History—Father of the patient was said to have been a dipsomaniac, while her mother died of tuberculosis. Nothing was known concerning the rest of her ancestors. Her only sister, however, was quite neurotic.

Personal History—Nothing unusual occurred in the life of the patient until about the fourteenth year, when she took a great dislike to one of her playmates, and after returning from school, where they met daily, she would wash her hands continually, and sometimes her gloves. She also showed signs of stubbornness, and was given to occasional hysterical outbursts. Her menstruation was irregular, but she seemed to be well otherwise. She was bright and intelligent, and quite precocious in her studies. Her general nervous condition, however, gradually increased in intensity as age advanced, and in addition to washing her hands constantly her ablution began to extend to other parts of her body. Upon being asked the reason for this marked desire to be cleanly, she stated that she feared she was being contaminated by her schoolmates and surroundings, and that she in turn was contaminating her own family.

Her general health remained fair, while her nervous condition was not considered of sufficient importance to need the services of a physician. Upon leaving school she studied stenography and soon secured a position, but did not remain long in any one situation as her peculiaities, such as constantly washing her hands, clothing, etc., attracted her employer's attention and she was dismissed. Her conversation was perfectly rational, and while recognizing the absurdity of her dread or fear of being contaminated she claimed she was wholly unable to refrain from the habit.

Associated with the fear of contamination she engendered a personal dislike for the article or person she thought contaminated her, which finally resulted in her tearing up her clothing or setting fire to the furniture of her room. Often in alighting from a train in some strange town or city she would be seized with the dread or fear that something had contaminated her, and no matter what inducements were offered, she would leave immediately for other parts; but before reaching her destination would stop at a hotel long enough to wash everything belonging to her. Nothing escaped—clothing, trunk, money, pocket-book, hat, shoes; indeed, everything she had with her. At one time she worked in a large western city, but soon became convinced that her associates in business, as well as her surroundings, had contaminated her, and soon after left for another city to visit friends. While there she learned that there were some people in the same house that had just arrived from the city in which she had formerly worked, which made her very uneasy and restless, and under no circumstances would she volunteer to meet the visitors, for fear they would contaminate her. Upon one occasion while her mother was entertaining some visitors at her own home, during a meal, in passing the food from one to the other the dress-sleeve of a visitor happened to touch the vegetable dish, which was sufficient to make Miss A. become much worried and miserable, until finally she left the table and went to her own room, where she stripped herself and took a sponge bath.

She would not write letters to persons who lived in cities where she had been employed, for fear that the reply would contaminate her, but when by chance she received such letters she would invariably wash them before reading! If while attending church she happened to see anyone she had formerly known in the cities where she thought she had been contaminated she would leave the church immediately for home and then wash all her possessions, as well as all the furniture in the house, until stopped by sheer force or until she became exhausted.

Often she takes a dislike to some article of clothing or furniture and immediately destroys it, and then bathes herself and all her belongings, including the bed, walls, pictures and doors, and very frequently will let certain articles of clothing, like her gloves or hat, remain soaking over night in water.

When restrained from using water she will pick and pull desperately at her clothing and other articles surrounding her, ever on a ceaseless hunt for the object or objects she believed contaminated her. When reasoned with she acknowledged that she is unreasonable, and that her fears are delusive in character, but cannot resist the impulse to either wash or destroy.

Upon her admission to the sanitarium she had been suffering from mysophobia for about ten years, resisting all efforts of herself and friends to overcome her ailment. For several years she had been under the observation of the family physician, who diagnosticated her condition as one of neurasthenia, but intellectually she was bright and rational, being perfectly conscious of her serious condition and voluntarily came to the sanitarium for treatment.

Examination revealed no organic disease of any kind, and her general health was fair. She ate and slept well, and took kindly to all the rules of the sanitarium. She did not complain of any pain, although her menses were irregular, and the only thing noticeable was a marked tendency to despondency. She presented a well-developed beard, of which she was very sensitive, but kept it closely shaven by using a razor daily. During her stay with us she sought at times every opportunity to carry out her impulse to wash or destroy, occasionally becoming quite deceptive, and even secretive, to accomplish this purpose. She was placed under rigid surveillance, and special attention was given to improvement of her mal-nourished condition. For this purpose tonics, electricity, massage, exercise, hydrotherapy and other agents were employed, including large amounts of nutritious food and psychotherapeutics. She gradually improved, and at the end of several months we thought we had fairly conquered her besetment, she herself claiming that she was entirely well and wanted to go home to spend the Christmas holidays. which request was also urged by her friends. She promised faithfully she would refrain from her morbid habits, but no sooner did she leave Kansas City than the old impulse returned, and she stopped on her way home at a small station, went to the hotel, secured a room and washed everything she possessed, then resumed her journey.

Soon after her arrival home she gave the entire premises a good scrubbing, including her own belongings; in fact, continued until she became thoroughly exhausted. About two weeks later she returned to the sanitarium unaccompanied by anyone, and after reaching Kansas City went at once to one of the hotels and had a general clean-up before coming to the sanitarium.

She regretted her conduct and the forfeiture of her pledge to me, but claimed she was unable to resist the desire to wash herself, money, clothing, etc. She also stated that the only persons she knew who never incited these impulses or caused the fear of contamination to arise in her were her two brothers, who seemed in her mind to be free from such power. Upon one occasion, in speaking to me of her love affairs she stated that while living in California she fell in love with a young man, but did not like to be with him very often, as she feared that should he make her angry the impulse to wash him, or herself, might suddenly arise, and nothing short of this would satisfy her. She is naturally affectionate and bore a most excellent character for kindness of disposition, yielding kindly and willingly to the dictates of friends, physician and nurse. Her improvement was so very satisfactory that on April 1, 1903, friends instructed me to send her to St. Louis to visit relatives. While there she seemed to get along very well for a few weeks, but relapsed into her old state, and finally became so troublesome that she was legally adjudged insane and sent to the State Hospital for the Insane at Topeka, Kansas, where she remained for some months, but finally was discharged improved. A few days ago I wrote her friends, to inquire after her present welfare, and they reported as follows:

"I consider L's condition much the same as when she left you. I don't think she is any worse, but she is certainly no better. The same idea of contamination seems to haunt her. She never leaves the house even for a drive, and seldom meets anyone who calls, for fear of being contaminated."

In analyzing this case I think we can all readily agree with Dr. Dewey[2] when he states in his able paper on "The Dividing Line Between the Neuroses and Psychoses" that so far as prevalent views are concerned, if there be a dividing line between the neuroses and psychoses insanity exists on both sides of this line, hence the term neurasthenia, or nervous prostration, is often a polite misnomer for insanity.

Dana[3] has also recently emphasized the close relation which exists between the various neuroses and psychoses in his paper entitled "The Passing of Neurasthenia." To use his own language, he says: It is my contention that a large number of the so-called neurasthenias and all the hysterias should be classed as prodromal stages. Abortive types or shadowy imitations of the great psychoses, for in these cases it is the morbid mind that dominates the situation, not a weak eye muscle or a poor stomach; a heavy womb, uric acid, arterial sclerosis, or even an exhausted motor nerve.

"They are not often, to be sure, pure psychoses, for the body is also at fault; but the *psyche* is in main control, and it gives the stamp to the clinical syndrome, directs the prognosis, and most acutely solicitates the treatment."

The practical clinical value and significance of the attitude thus assumed by Dr. Dana cannot fail to appeal to all of us, while his claims are amply sustained and justified by my own college and hospital clinical experience, as well as by the case record of those admitted to my sanitarium. It is the rarest exception in our admission to find a pure, unadulterated case of neurasthenia or hysteria, although so diagnosticated by the family physician.

The vast majority, however, said to be suffering from these nervous affections when admitted are, upon strict examination, found to be afflicted with a true psycho-neurosis, which we sometimes designate as psychasthenia, or which perhaps could more properly be termed *psychosomatasthenia.*

While the so-called neurasthenias and hysterias may not conform to the legal test of insanity, yet it is clear that the large majority of persons so diagnosticated are not endowed with normal minds, and that their maladies or sufferings are largely due to the mismanagement of their mental faculties rather than their bodies, hence the morbid mentalization constitutes the chief pathological element of their ailment.

Recognizing, however, that associated with the psychical manifestations of these conditions there are also commonly found gross functional and even serious organic somatic changes, requiring possibly at times surgical interference for their relief, yet we contend that even when these purely local bodily complications have been fully met they often fail to relieve, much less cure the mental agony of the patient.

This would therefore indicate that in these morbid neurotic states we are dealing essentially and primarily with a true psychosis, and in order to treat them successfully we must of necessity apply the same rules or principles as those which belong to any other form of incipient insanity. Failure, however, to recognize the expedience of such stringent measures is largely the responsible agent for much of our present inability to cure such ailments. This discrepancy, therefore, forms the very climax of my theme, for if there be one principle more than another that has been universally endorsed and emphasized by you for the past quarter of a century in your biennial reports, and to my mind justly so, it is the fact that insanity is more curable in its incipiency than at any other time. The longer its duration without appropriate treatment the less the chance of recovery. Every alienist and neurologist responds to the logic of this dictum, for its truth is so very apparent that it can readily be demonstrated by actual practical clinical experience, and although hampered at times with the baleful influence of a tainted heredity, yet it is the crowning glory of modern medical science to possess the means of overcoming this great congenital bugaboo.

If our united experience warrants the assertion that insanity in its incipiency is extremely curable, why allow it to become incurable before applying the legitimate means and measures that favor its cure?

We are all, however, compelled to admit that the practical application of the great law *prevention*, as related to so-called neurasthenia, hysteria and allied states, is a very difficult one, even in spite of our united favorable opinion of their incipient curability.

Herein, however, lies, in my judgment, the special responsibility of the medical profession, for as alienists and neurologists we must of necessity continue to sound the alarm in the consciousness of our own appropriate treatment, and in the presence of universal mental and physical wreckage and distress which results from error or neglect of its judicious adoption.

While the morbid psychological manifestations in this case were present as early as the fourteenth year, yet practically nothing of a tangible therapeutical character was done for their relief until the twenty-fifty year, at which time their chronic fixity rendered the prognosis most unfavorable. It is, however, only reasonable to infer that had all the resources of modern medical science been brought into requisition early, and the principles which apply to the treatment of incipient insanity been rigidly enforced, the possibility of a cure would have been greatly enhanced.

If the present percentage of recoveries does not exceed 30 per cent., and there is universal agreement that the rate of recoveries could be raised, according to different authorities, from 75 to 90 per cent., provided all the resources of modern medical science were brought into requisition in their incipiency, it would seem our imperative duty to demand such legal and medical reforms as are necessary to meet this desirable exigency.

Kirchoff[4] has clearly shown that certain insanities, such as melancholia, mania, paranoia and general paresis, may all develop upon a neurasthenic basis. While Chapin,[5] in his work on insanity, declares that "the larger proportion of admissions to the Pennsylvania Hospital received in an acute stage of insanity give a history of neurasthenia. He therefore fitly styles it "the soil out of which insanity develops."

That there is, however, a marked difference between true neurasthenia and insanity all will agree, but at present the differentiation of the various psychological manifestations which complicate neurasthenia and their separation into independent morbid entities are not well defined; but it is safe to assume that the most important clinical sign of neurasthenia developing into a true mental disorder is the manner in which the feelings, thoughts and actions of the individual absorb his entire attention. This self-consciousness or morbid introspection is a marked feature of the more serious aspects of neurasthenia, and simply betrays the serious invasion of the higher mental faculties in the progress of the malady. Moreover, this neurotic weakness makes the patient vulnerable to morbid impressions of the ego, which is usually expressed, clinically, in the form of anxiety, distrust or suspicion. These morbid concepts have been variously designated by different authors as obsessions, besetments, impulsions and fixed ideas; but clinically they are expressed either in the form of doubts, fears or impulsive acts, which irresistibly force themselves upon the individual, dominating his every thought, word and deed. In a limited degree they are commonly present in health; indeed, very few of us escape the presence of doubts, fears or impulses, as isolated sudden thoughts, but these are usually subject to the dominating power of inhibition. When they appear, however, as a complication of neurasthenia they often get beyond the control of the will and may, therefore, constitute the chief factor of a progressive insanity. Moreover, they are ably shown by Regis to be due to lesions of the will or the offspring of a morbid heredity, hence there can be little doubt that certain crimes are due to such obsessions, impulsions and nosophobic impulses, which may suddenly arise in the mind of such who are not ordinarily regarded as insane, but whose responsibility should always be duly considered. While they present themselves clinically in every degree of intensity, yet it is the degree combined with their persistency which indicates their true seriousness.

In conclusion, it would seem from what has been said that we are justified in formulating the following deductions:

First—That the close relation which exists between the so-called neurasthenias and insanity is so very striking as to establish a true equivalency.

Second—That curability of these insanities largely depends upon the early enforcement of appropriate medical treatment.

Third—That the non-insane psychoses are subject to the same therapeutic laws and principles which apply to the more pronounced forms of incipient insanity.

Fourth—That their prognosis is in direct ratio to their duration.

Fifth—That violation either through ignorance, pride or wilful neglect of these fundamental essentials is the responsible agent of the rapidly increasing tide of incurable mental disorders.

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[5] "A Compendium of Insanity."

ACUTE ANTERIOR POLIOMYELITIS IN A YOUTH.

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(*Continued from page* 557.)

The anterior root zone shows numerous blackened areas by the Marchi method, a scarcity of nerve fibers and a beginning sclerosis. This degeneration is less marked in the lateral columns. In the external part of the posterior columns there is a diffuse, though well-marked area of degeneration which is rather more sharply defined in the dorsal region. The white matter below the fourth lumbar segment does not appear unusual though the gray matter still contains small areas of destruction.

Etiology.—There is a very wide range in the etiology of this disease, varying from slight colds to the most severe infectious diseases. Age, season, trauma and infectious diseases are the most important etiological factors. The average age of onset is 3 years. Most cases occur under 10, and four-fifths of these under 3. It may, however, begin as late as 60, but most adult cases appear before 30. It will be seen that by far the greatest number of cases develop at the age when the child learns to walk; that is, when the spinal centres are unusually active and consequently more susceptible to toxic and other accidental causes which may be present. It is in the hot summer months when the disease finds most of its victims, more especially from June to September. Most of the large epidemics have occurred during these months.

Trauma.—Traumatic influences are perhaps more potent than a hasty review of the literature would lead us to believe. Several cases have been reported which followed falls and injuries directly. Bullen[7] reported a case of paralysis of both legs. Two years later a weight fell upon the patient's left hand which soon became paralyzed.

Two cases of traumatic origin are described by von Leyden[28]; one in a man, æt. 63, whose leg was fractured at the age of 2, followed by arrested development of the leg. A sclerotic lesion was found in the gray matter of the cervical cord. In the second case, paralysis of the leg followed an injury to the foot.

Stark[58] mentions the case of a woman who fell from a wagon, striking on her back and right shoulder. The injury was soon followed by weakness of the right shoulder and arm which terminated in paralysis. The anatomical changes in this case were characteristic of poliomyelitis with pronounced parenchymatous changes, but only slight evidence of inflammation. A case of injury to the arms, followed 6 months later by progressive weakness of the right hand and arm and a diffuse amyotrophy with reaction of degeneration is cited by Perrin.[40] One year later the left arm became involved, and finally the trunk and legs.

Burckhardt[8] reports a case of a boy, 1 year old, whose face was scalded with boiling water. He developed vomiting and fever in a few days, with paralysis of all the extremities and trunk muscles. He died four days after the accident, and the cord showed macroscopical and microscopical changes in the gray matter.

E. Meyer[31] observed a progressing paralysis of the right leg following injury to the right foot in a man 59 years of age. Paralysis of the left leg soon followed.

A case of radial paralysis with subsequent paralysis of the arms soon after delivery is reported by Gumpertz.[28]

Flatley[18] considers his case of anterior poliomyelitis in a man 27 years of age, who fell from a wagon and three days later developed a flaccid paralysis of the left upper arm, associated with Bell's paralysis and syringomyelic sensory dissociation, of traumatic origin.

Decroly[14] questions whether the fall caused the paralysis or *vice versa*, in two cases of supposedly traumatic origin.

Epidemics.—That the disease is probably of an infectious nature has become widely accepted. Its development and course remind one of an infectious disease. Several distinct epidemics have been reported which have been so widespread that the factor of a mere coincidence can be ruled out without a question.

Caverly[9] recorded a remarkable epidemic which occurred near Rutland (Vermont). During the summer of 1894, 119 cases developed. Eighty-five were under 6 years of age, and 18 died. Macphail[29] described the same epidemic.

A Stockholm physician, Medin[30] has reported an epidemic in which 44 cases occurred in one month in the same town.

In 1888 Rissler[46] had an opportunity of studying the pathological changes occurring in the cord in 5 cases during an epidemic. Leegard[25] narrates a case of an 8-year old boy who developed anterior poliomyelitis; four days later a 20-year old servant came down with the same disease.

Johannessen[24] gives a list of the number of cases occurring in Stockholm from 1888 to 1895. They varied from 3 to 11 in a year, except in 1895, when there were 21 cases—4 in July, 4 in August, 8 in September, 2 in October, 2 in March and 1 in June. In 1889 there were 5 cases in Jena—1 in June and 4 in July; age from 9 months to 3 years.

Pierracini[42] reported an epidemic of 7 children in 15 days in a town in Italy near Florence. Bucelli[6] saw 17 cases within four months.

Auerbach[2] had 15 cases from May to December, 1898; 4 on the same street. He goes as far as to say that the difference between epidemic poliomyelitis, cerebro-spinal meningitis, sporadic or epidemic, and encephalitis, is not one of pathogenesis but merely that of selection of the same micro-organism, the Weichselbaum-Jaeger meningococcus.

Johannessen[24] studied 23 cases from 1893 to 1899, 16 boys and 7 girls. Two cases occurred from March to May; 10 from June to August; 5 from September to November, and 1 from December to February. He thinks that it is possible that some relation exists between poliomyelitis and the gastro-intestinal disturbances to which small children are subject. He quotes Bülow-Hanson and Harbitz, who found swollen mesentary glands and hyperemic Peyer's patches.

In an epidemic of poliomyelitis, Leegard[25] observed 54 cases: 45 of these occurred from July to October; 12 had gastric symptoms. Colds were given as causes in 6 cases; exertion in 7 and both in 9. Heredity seemed to play no rôle. In two districts there was also an icterus epidemic. The epidemic nature of the disease was shown in the fact that it spread along the lines of most extensive travel.

A remarkable family epidemic in which all of 7 children were attacked within 10 days was recorded by Pasteur.[39] Two recovered completely; the others had residuals.

Painter[38] had an opportunity to study an epidemic of 38 cases which occurred in Gloucester (Mass.) within a radius of 4 miles, and most of them near together. This epidemic lasted from June to September.

Colmer[12] reported 8 or 10 cases of teething paralysis, identical with anterior poliomyelitis in West Feliciana (La.).

The following cases are cited by Painter, but no references are given. In 1881 Bergenholtz observed 13 cases in 1½ months. Oxholm reported 9 cases in a small community in Norway in an interval of 6 weeks. Cordier described 13 cases near Lyons (France) in June and July, 1885. In 1896 Brackett reported an epidemic of 8 or 10 cases in North Adams (Mass.). In 1899 5 cases in a population of 49 were observed by Newark. Two fatal cases of cerebro-spinal meningitis occurred during the epidemic reported by Painter. No relation between the cases of anterior poliomyelitis could be established but the symptom-complex in all was very similar. Some cases began during very hot weather; others after a sudden drop of temperature and others after bathing. One case died. The ages of the patients varied from 15 months to 10 years. Eight cases, 2 years or under; 21 cases, 3 years or under; 7 cases, 4 years or older.

Packard[37] reports a case in which two sisters were attacked within 3 days.

J. M. Taylor[56] saw an epidemic in a village of 1,500; 4 cases were seen, 2 being brothers. Two other cases occurred in a neighboring town.

A similar case, in which a 4½-year-old boy developed anterior poliomyelitis one week before his 3-year-old sister, is cited by L. Guinon and Rist.[22]

Lepine[26] observed a case of typhoid myelitis occurring in the course of typhoid fever and probably caused by the bacillus of Eberth. Typical lesions of anterior poliomyelitis were found in the lumbo-sacral cord. The cerebro-spinal fluid was increased in quantity.

According to Gossage[21] the disease attacks strong as well as weak children, and often follows the acute specific diseases of childhood. Convulsions and coma are common initial symptoms in epidemics, but rare in sporadic cases. In Medin's epidemic, two cases of multiple neuritis occurred at the same time with anterior poliomyelitis. The relation of neuritis with anterior poliomyelitis has also been observed by other writers.

An epidemic of neuritis in which two cases of anterior poliomyelitis occurred came under the observation of Eisenlohr.[16]

Bacteriology.—The highly suggestive clinical course of the disease, together with the numerous epidemics which have been reported, have prompted some of the more recent investigators to make bacteriological examinations of the cord and cerebrospinal fluid, while others have attempted to produce the disease experimentally in animals. Both have been partially successful, though a uniformity in results is still lacking. There is as yet no proof that the disease depends upon the action of some specific micro-organism, but sufficient evidence has been accumulated to show that it is dependent upon toxins, of various kinds, either bacterial or other poisons.

Schultze[49] found in one case the Weichselbaum-Jaeger meningococcus by lumbar puncture, and believes the coincidence with meningitis is far from rare.

Dercum[15] made a lumbar puncture in a case of anterior poliomyelitis and found numerous micrococci which morphologically and tinctorially resembled the diplococcus of Sternberg.

Spiller[52] showed the presence of the staphylococcus pyogenes albus in cultures made from the intradural fluid of the cord in a case of paraplegia, resembling transverse myelitis; but it showed at autopsy an extensive poliomyelitis.

Engel[17] made a lumbar puncture in a case of anterior poliomyelitis six days after the onset of the paralysis in a 5-year-old child. No micro-organisms were found in the clear fluid, but cultures showed the presence of the staphylococcus pyogenes albus (patient had had purulent otitis media since the age of 16 weeks).

Chapin[10] described an epidemic in which Dr. Brooks found diplococci in the anterior horns, but not in the blood or meninges. In a second case diplococci were found in the blood during life.

H. Vincent[58] isolated the Eberth bacillus and another one strongly pathogenic for rabbits, from the spleen of a patient who died of typhoid fever. He inoculated several rabbits with a mixture of this culture, and produced a severe enteritis. After this improved, he noticed a rapid paralysis beginning in the hind legs. The animal died 14 days after the onset of the paralytic symptoms. The autopsy showed typical lesions of anterior poliomyelitis in the lumbar cord, absence of cells and thickened vessels. He was, however, unable to produce it at will in other animals. He was unable to isolate the bacilli from the rabbit, and considers the lesion the result of the absorbed toxins from the bacteria and not resulting directly from their presence.

Roger and Bourges[47] produced similar lesions after injecting streptococci. Guinon and Rist[22] made lumbar punctures in the cases referred to above, 14 days after the onset in the boy, and 7 days after the onset in the girl. The cerebro-spinal fluid was sterile and without leukocytes.

Thomas[57] reports two cases of acute ascending paralysis which, however, were considered anatomically as acute anterior poliomyelitis. Bacteriological examination of the sections and cultures were negative. A negative result was also obtained in Bülow-Hansen and Harbitz's case.

Batten[4] made bacteriological examinations of the cerebro-spinal fluid and of the anterior horns of a case of anterior poliomyelitis and found in the cerebro-spinal fluid: (a) a short difficult staining bacillus (probably proteus vulgaris); (b) a fat coccus resembling cocci from infected wounds; and (c) many ordinary staphylococci. From the anterior horns he isolated: (a) a short poorly-staining bacillus, but not the same as the other: (b) the same fat coccus but no staphylococci.

In a recent case six weeks after the onset Achard and Grenet[1] found a pronounced lymphocytosis in the cerebro-spinal fluid of a 14-year-old child suffering from anterior poliomyelitis.

In contrast with this observation J. Fraenkel[19] and Niedner and Mamlock[38] found no lymphocytosis in cases of poliomyelitis.

The peculiar course which the disease took in this case reminds one strongly of an acute ascending paralysis. Cases have been reported clinically as Landry's paralysis which at autopsy have shown lesions characteristic of anterior poliomyelitis and should really be classed in this group. Clinically the two conditions are at times inseparable and no hard and fast lines can be drawn between them. With a rapid fatal termination and development of bulbar symptoms, we speak of it as Landry's paralysis. Both the clinical and anatomical findings of the two diseases overlap to such an extent that the too free use of the term "Landry's paralysis" is very confusing. Acute ascending paralysis seems to be more the result of a toxic or infectious process. In many cases splenic enlargement, inflammation of the lymph glands, and albuminuria were noticed. As in poliomyelitis various micro-organisms have been described in the blood and organs.

Bassoe[3] reports a case of acute ascending paralysis in which the lesion was a hemorrhage poliomyelitis. Encapsulated diplococci were isolated from the heart's blood. The bacillus coli communis was found in the cord, spleen and peritoneal fluid. In the liver a pneumococcus and the staphylococcus cereus flavus were found. He also produced experimentally a tigrolysis of the ventral horn cells in a rabbit inoculated with the staphylococcus pyogenes albus. He quotes Mönckeberg, who also found a hemorrhagic poliomyelitis in a typical case of Landry's paralysis.

Pathology.—While the literature of poliomyelitis is very extensive and the disease is one pointing to definite and easily recognized changes in the spinal cord, more particularly the gray matter, there are still some points which are by no means clear. The etiological factors are very uncertain and obscure. The reason why the cord alone should be attacked, and just how the infection or intoxication reaches the cord, is in doubt, and it remains a mooted question whether the disease attacks primarily the nervous elements or the vessels.

There are still many who adhere to the teachings of Charcot[11] that the disease is primarily of a parenchymatous nature, in spite of the many strong arguments in favor of a primary vascular change. Charcot made his examinations in cases of long standing after the inflammatory condition had subsided, which explains in part the reason for his conclusions. His was the generally accepted theory until Roger and Damaschino[48] had an opportunity of studying several recent cases from which they concluded that the vascular lesions were probably primary.

Bruining[5] is one of the few recent writers who adheres to Charcot's views. He studied clinically two interesting cases of father and son in whom the disease began in adult life. The father died two years after the onset and the cord showed extensive atrophy of the anterior horn cells. The vessels were congested but there was no perivascular infiltration. Numerous large neuroglia cells, with protoplasm staining red with eosin, were seen in the anterior horns of the dorsal cord. From the absence of any inflammatory changes, the author concludes that the disease attacks the nervous elements primarily.

Bullen[7] reports an adult case in which the pathological appearances were for the most part those of parenchymatous atrophy only.

Nonne[34] likewise considers the cell destruction in the anterior horns as primary. No vascular changes were found in his case, but the examination was made two years after the onset. A few writers (Prætorius[44], Schwalbe[50], Preobrajensky [46], and others) are still uncertain whether the toxin affects vessels or cells primarily, or both. They incline, however, toward a primary vascular involvement. Most of the modern writers take a definite stand in favor of primary interstitial changes and secondary involvement of the nervous elements. Among them may be mentioned Siemerling[51], Dauber[18], Goldscheider[20], Placzek[48], Taylor[55], Mott[32], and Batten[4].

Let us consider for a moment the pathological-anatomical characteristics of the cord in poliomyelitis. The general structure of the gray matter is greatly altered; the vessels are dilated and there is enormous distension of the perivascular spaces with cells of various kinds. There is hyperplasia and hypertrophy of the neuroglia cells and scar tissue is abundant in the subacute and chronic stages. This alteration in structure of the interstitial tissue is sufficient in itself to exert a strong traumatic influence on the nerve cells. Large masses of extravasated cells, and even small hemorrhages, may be seen at times in close contact with the ganglion cells. These cells are as a rule smaller than normal; their prolongations cannot be made out and the chromophilic granules are closely packed together; the cells appear flattened, and cut off as if by pressure. The grade of cell destruction seems to depend on the proximity of the cell to the most extensive interstitial changes. A few cells show a central chromatolysis. They are, however, not as common as the former and in this case only cells in Clarke's column show this tendency. Taylor[55], Siemerling[31], and others speak rather in favor of the mechanical action of the interstitial alterations upon the nerve cells than for toxic influence upon them. It seems not improbable that in the early acute stages the cells may be acted upon by the poisons simultaneously with the vessels; but pathological studies of such cases do not bear this out entirely. The cord shows either a more or less widespread hemorrhagic condition or the presence of a violent inflammation involving more particularly the vessels without any visible structural change of the nerve cells. The widespread scar tissue in the gray matter of some acute cases, such as in the present one, with here and there a fragmented cell, makes one think that the destruction of these cells certainly depends largely on the pressure.

In view of the fact that anterior poliomyelitis may be the result of various poisons, we are hardly justified in attempting to overthrow the theory of a primary parenchymatous involvement entirely, since we know that such poisons as lead, arsenic, etc., are able to produce distinct changes in the cells of the central nervous system. Pathological alterations, typical of anterior poliomyelitis, have been demonstrated in poisoning of various kinds, more especially by lead. These cases might at first lead one to suppose that the nerve cells have a marked affinity for lead and were primarily destroyed by its direct toxic action. However, according to the observations of many, lead causes intense vascular changes which lead to impairment of blood supply and secondary cell destruction. Onuf[36] reports a case of a painter in whom there developed complete flaccid paralysis of both lower extremities, with absent knee jerks. The autopsy revealed probable poliomyelitis of the anterior horns, enormous infiltration of round cells about the vessels, general destruction of the nerve cells and marked involvement of the anterior roots. In experiments with lead on 36 animals, Stieglitz[54] obtained in one guinea pig an acute poliomyelitis with cell infiltration and more or less destruction of nerve cells around the foci. This guinea pig had been paralyzed acutely and death occurred within 24 hours. Vulpian[59] found a pronounced poliomyelitis with colloid degeneration and atrophy of the cells in the case of lead poisoning. Small-celled infiltration and sclerotic islands were present in the roots of the cervical enlargement. He also produced artificial lead intoxication in a dog with paralysis of the extremities and demonstrated an exquisite poliomyelitis. A. Westphal[60] and others agree that the lead acts primarily on the central nervous system, but in its selective action it varies. It may attack the nervous elements, or the vessels, or both. Philippe and Gauthard[41] report a case of anterior poliomyelitis resulting from lead poisoning, and Obrastzoff[35] demonstrated anterior poliomyelitis following arsenical poisoning. Poliomyelitis has occurred during the course of typhoid fever (Lepine), and Leri and Wilson[27] attribute the poliomyelitis in their case to syphilis.

A very important factor in determining the peculiar susceptibility of the cord to various disease processes is its blood supply. The distribution and extent of the inflammatory process seems to depend upon the arterial supply of the gray matter. The gray matter receives its greatest nutrition from the branches of the anterior spinal artery. It divides into two main trunks within the gray matter each of which is again subdivided into an anterior, a median and a posterior branch. In addition, a number of large and constant vessels enter the gray matter from the pia. The most important of these are the vessels entering with the anterior and posterior roots (the anterior and posterior root arteries). Another very constant vessel passes from the pia to the gray matter at the junction of the anterior and lateral columns (antero-lateral artery). Numerous smaller vessels also enter the gray matter from the pia. The anterior and posterior spinal arteries are derived from the vertebrals and are reinforced by branches of the intercostal, lumbar and other arteries. These vessels form transverse anastomoses in the pia mater and pass into the gray and white substance of the cord. Some of the peripheral or pial vessels simply form superficial capillary loops; others are distributed to the white matter. Most of them penetrate into the gray matter and supply the parts not served by the branches of the anterior spinal. A series of small arterioles enter the posterior fissure and give off branches which supply the adjacent parts of the posterior white columns and Clarke's column, where this is found. No one part, however, is supplied by one set of arterioles, nor is any one set of arterioles confined in its distribution to any one white column or group of cells in the gray matter. Within the cord, as within the brain, all the arteries are terminal. They do not anastomose with other arteries, but each one terminates in its own capillary area and supplies no other. The gray matter of the anterior horn is at the extremity of these vessels and the collateral circulation is poor. This factor is certainly of great importance since it offers a favorable field for sluggish circulation and embolism. It has been suggested by some writers that the lumbar and sacral cords are affected so often, because the blood must travel throughout the length of the anterior spinal artery from the vertebrals, apparently disregarding the fact that these parts are reinforced by branches of other vessels.

Various groups of cells at different levels are diseased indiscriminately and do not follow any definite system. If but one group of vessels, the anterior spinal, were diseased, we might consider the variability of the distribution of the lesions to depend upon the irregular distribution of branches of this particular vessel. However, as specimens of this case show, the peripheral vessels are equally if not more constantly involved than the branches of the anterior spinal arteries and areas of anemic necrosis, small hemorrhages and localized destruction of cells, can be seen in the areas served by these vessels, while the parts supplied by the anterior spinal artery are but little involved. This patchy involvement is present in the cervical and lumbar regions only. One very striking phenomenon which the sections of this case show at every level is the fact that the diseased vessel, whether of central origin (anterior spinal) or penetrating through the white matter into the gray matter, shows the perivascular spaces to be infiltrated with cells practically only in the gray matter. If present in the white matter, the infiltration is of slight extent or in the immediate vicinity of the gray matter. Not a single specimen shows any perivascular infiltration about the anterior spinal artery until its branches have reached the gray matter. This helps to emphasize the selective action of the disease primarily on the gray matter of the cord, and the loose physical architecture of the gray matter probably determines this to some extent. In the areas of most pronounced disintegration of the anterior horn the small-celled infiltration is most extensive, in some places leading to small hemorrhages and in others to large masses of lymphocytes closely packed together, but nowhere to actual pus formation.

From the present case it appears that owing to the anatomical peculiarities of the gray matter, it is the gray matter as a whole which is destroyed and with it the nerve cell. The case demonstrates, I believe, that the gray matter of the cord receives rather more nutrition from the peripheral vessels than is generally taught, especially in the antero-lateral regions. The disease, likewise, involves the peripheral vessels to the same extent that it does the anterior spinal artery. Preobrajensky[46] also found the antero-lateral artery diseased.

The views now generally held, that poliomyelitis is primarily due to a diseased condition of the blood vessels, have been materially strengthened by comparatively recent studies, revealing the presence of thrombosis and embolism in the spinal arteries. These observations represent a decided advance in the elucidation of the pathology of poliomyelitis, especially since we know that the collateral circulation within the cord is established with difficulty, and the presence of a thrombus or embolus in one of the smaller branches of the spinal vessels would shut off the blood completely from the part supplied by it. Mott[32] found inflammatory thrombotic stasis of the terminal branches of the anterior spinal artery, ending in flask-like areas of softening. Batten[4] also found a thrombosis of the anterior spinal artery. It has been demonstrated experimentally that occlusion of the spinal arteries produces a chromatolysis of the anterior horn cells, but inflammatory changes of the vessels are absent. Although a thrombosis in anterior poliomyelitis may be primary, the cell changes which are more extensive must depend not only on the cutting off of the blood supply but also upon other factors such as mechanical pressure and toxins.

From the present case, and from a review of the literature on this subject, we may be led to summarize as follows:

1. Anterior poliomyelitis is the result of a primary inflammatory disease of the blood vessels of the cord which may be thrombotic or embolic.
2. The destruction of the ganglion cells is secondary and depends in part upon the deficient blood supply of the diseased area and in part upon pressure and toxins.
3. The pathological changes occurring in poliomyelitis of children and adults are apparently identical and dependent on similar causes.
4. There is sufficient evidence at hand to consider the disease as a rule of an infectious nature, however, not depending upon a specific micro-organism but resulting from bacterial infections of various kinds, and at times from other poisons.
5. The inflammatory changes are present in the peripheral vessels as well as in the branches of the anterior spinal artery, though these changes are seldom visible until the vessels enter the gray matter.
6. The inadequate collateral circulation within the anterior horns is favorable for sluggish circulation and embolism.

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ANESTHESIA ASSOCIATED WITH HYPERALGESIA SHARPLY CONFINED TO AREOLA-NIPPLE AREA OF BOTH BREASTS: A NEW AND APPARENTLY CONSTANT STIGMA IN HYSTERIA.

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"Hysteria is a Proteus, assuming many forms, and like the chameleon, shows many colors."—Sydenham. Almost every bodily function may be perverted and organic disease is often so closely simulated by it, that the possibility of hysteria must be frequently reckoned with in neurological examinations. Inconstancy and variability in symptomatology are the chief characteristics of the disease.

Of the subjective symptoms—pain and paresthesia and of the objective symptoms—anesthesia and analgesia are the most constant. Oppenheim states, that pain is never absent in hysteria, and Gendrin was of the opinion that "In every case of hysteria, without exception, from the beginning to the termination of the malady, there exists a condition of anesthesia, general or partial." In four hundred cases observed by Briquet, some degree of anesthesia was found in two hundred and forty, or in sixty per cent. In a more recent series of forty cases, Pitres found sensation to be normal in two, or in only five per cent.

The views of Gendrin have never been fully accepted, for the reason, that it was not uncommon, especially in the interparoxysmal stage, for one to be unable to determine a condition of anesthesia. Anesthesia, general or partial, has, therefore, up to the present time, not been considered a constant symptom of hysteria. The same may be said of the so-called stigmata, and of these, corneal and pharyngeal anesthesia and *ovarie* are the most constant; but these may be present in otherwise normal individuals and they are often absent even in well-marked cases of hysteria, and hence are of little value in differential diagnosis. Certain visual anomalies and the characteristic sensory disturbances, when present, are pathognomonic of hysteria, but the former is less constant than the latter, and both are often absent. Even the hysterical temperament or mental state is not always sharply defined. Thus it is, that inconstancy and variability are expressive of all hysterical symptoms and stigmata, thus far recognized, and are equally expressive of the disposition and the mental state of the hysterical individual.

I have recently observed a peculiar sensory disturbance nowhere recorded in neurological literature, and one which is apparently constant in hysteria.

About one year ago, while making a sensory examination of a female aged forty-two, whom I had known for many years, and had frequently attended in hysterical fits, and in divers other hysterical manifestations, the only objective sensory disturbance found was anesthesia associated with hyperalgesia sharply confined to the areola-nipple area of both breasts. The tests were made with a fine wisp of absorbent cotton, and the pointed end of a rather large safety pin. Touch and pin-pricks were promptly recognized and accurately localized throughout, excepting within the areola-nipple areas, where touch was not recognized, but pin-pricks were said to be, and appeared to be more painful than to the adjacent skin. The anesthetic and hyperalgesic area was separated from the normal with hair line sharpness—and this line was represented by the skin-areola margin. Within the hyperpigmented area anesthesia and hyperalgesia were present—beyond it normal sensation.

Such findings were unknown to me and little importance was attached to these at the time, other than to consider them as objective evidence of the malady in question in this particular patient. Nevertheless, I was so impressed with the findings that I have made it a rule since then, in both private and clinic work to note especially the state of sensation of the areola-nipple areas in every individual coming under my observation. I have also made similar examinations in quite a number of healthy and apparently normal individuals of both sexes, varying in age from six to eighty years. My observations thus far have shown: (1) That in no normal individual was this sensory disturbance found; on the contrary, the majority of these claimed to perceive touch more, and painful impressions less acutely in the areola-nipple area than in the surrounding parts, whereas others noticed little or no difference. (2) That it was not present in any organic disease, neurosis or psychosis unless hysteria was a complication and the diagnosis of such complication was made independently of it. (3) That in every case of hysteria, thirty in all, six males and twenty-four females, including the mildest and severest examples of the malady, two of the males having traumatic hysteria, areola-nipple anesthesia associated with hyperalgesia was invariably present, conforming in a striking manner to my first observation recited above.

The presence of areola-nipple anesthesia associated with hyperalgesia was confirmed by my colleagues Drs. Frank R. Fry, and Sidney I. Schwab, in several of the above-mentioned cases, and also observed in several of their own; otherwise, I should have greatly hesitated to give it publicity at this early date. When, however, others are able to confirm one's observations, self-deception is to a certain extent eliminated, but the diagnostic value of an observation can only be determined by time and in the crucial balance of general experience.

My own experience compels me to consider areola-nipple anesthesia associated with hyperalgesia as a pathognomonic and practically constant stigma in hysteria, and should the observations of others prove confirmatory of its presence and constancy an additional objective diagnostic aid will be at our command, whereby we may be better enabled to differentiate between the so-called functional disorders, and between the hysterical and the organic.

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February 7, 1905.

The President, DR. JOSEPH FRAENKEL, in the Chair.

Two Cases of Dysbasia Angiosclerotica (Intermittent Claudication), with Demonstration of Calcareous Deposits in the Pedal Arteries.—Dr. J. Ramsay Hunt read this paper, and the demonstration of radiographs was by Dr. Arthur Holding. The first patient was a man, 49 years old. His occupation for the past 32 years had been that of an iceman, which necessitated long and frequent exposure of the feet to cold and wet. The patient had used alcohol and tobacco to excess for many years. He acquired syphilis in 1879. The following year he had some ulcerations on the right leg, and five years later suffered from ulceration of the palate, with perforation. In September, 1903, he had an attack of delirium tremens, for which he was treated in the alcoholic pavilion of Bellevue Hospital. Restraint was necessary, and he was bound in the usual manner by sheets attached to the wrists and ankles. The first symptoms of the present affection appeared immediately after his discharge from the hospital, and was attributed by the patient to the restraint used. He complained of stiffness in the toes and the left leg; also the ankle, and paresthesia of the toes and edges of the feet. After walking a quarter of a mile the prickling, pain and stiffness became so distressing that he had to sit down until it wore off, which was the case after a few minutes' rest. His symptoms were much worse in winter than in summer, and were especially severe on a cold day. On rising in the morning the feet and legs felt perfectly well and natural. Toward the end of the day the feet became puffed up and swollen, and presented a mottled and cyanotic hue. He complained that they felt cold most of the time. The posterior tibial artery was not palpable on either side. The dorsalis pedis was full and larger than normal. The femorals and popliteals pulsated normally on the two sides. The feet were cold, and on standing became red and swollen, with a distinct purplish tinge. Numerous venules and minute venous varicosities were present about both ankles and along the anterior tibial region. The toe-nails on the left foot presented transverse ridges. The reflexes and sensations of the lower extremities were normal. Signs of a general arteriosclerosis were present, and the urine contained a faint trace of albumin, but no casts nor sugar. Its specific gravity was 1.017. Subsequent examination revealed an occasional faint pulsation in both posterior tibials, weaker on the left side.

The second patient presented by Dr. Hunt was a man 60 years old, who for fifteen years had been a longshoreman. For the past ten years he had been exposed a good deal to the cold and wet. He denied lues. For the past fifteen years he had smoked from ten to fifteen cigars daily, and had used alcohol to excess. About five years ago he began to suffer from numbness, stiffness and cold sensation in the toes of the right foot, coming on about two hours after beginning work in the morning. At the end of the first year he was compelled to rest after about three hours. At the end of two years his condition had grown worse; the paresthesia and stiffness had extended as high as the ankle, and rest was necessary after two hours' work. After resting, and in the morning on rising, all his symptoms had disappeared. The symptoms in the left foot gradually grew worse, and six months ago similar symptoms appeared in the right foot, beginning in the toes. Both feet assumed a mottled appearance on standing, but did not swell. A pulsation of the femorals, popliteals and posterior tibials could be felt on both sides, while the pulsation of the dorsalis pedis was entirely absent on both sides, although the artery could be felt as a fine-rounded cord. The reflexes and sensations of the lower extremities were normal. There were well-marked signs of a generalized arteriosclerosis. The urine had a specific gravity of 1.023; it contained a trace of albumin, no sugar. The symptoms presented by the cases shown were to be ascribed to an arteriosclerosis of the vessels of the leg and foot—the so-called dysbasia angiosclerotica (intermittent claudication of Charcot). The X-ray photographs demonstrated by Dr. Holding showed very exquisitely deposits of lime in the posterior and anterior tibial arteries and the dorsalis pedis, marking out very definitely the course of the vessels. As might be inferred from the general evidences of arteriosclerosis in both cases, the pathological process in the pedal arteries was the usual arteriosclerosis of Gull and Sutton, with deposits of lime in the media. These cases are supposed to be less amenable to treatment than the endarteritis forms; hence this method of demonstration might have a certain prognostic value. Both cases had shown a very moderate response to treatment.

Dr. B. Sachs called attention to the similarity of the lesions met with in this condition to those of erythromelalgia.

A Case of Probable Infectious Myelitis of the Cervical Region. Following a Compound Fracture of the Jaw.—This was also presented by Dr. Hunt. The patient was a man 36 years old, a painter by occupation, who, until the onset of the present trouble, had enjoyed excellent health. He had never shown any symptoms of lead poisoning. On Dec. 7, 1904, he fell on the street and fractured his lower jaw in two places. The fracture was a compound one, and the wound, which was on the inside of the mouth, bled for several hours. Three weeks later he had rigors at night, on one occasion followed by profuse perspiration. On Jan. 5, 1905, after a heavy slumber, he awoke with severe pain in both arms, which were also very weak, so that he could hardly lift them. The weakness was chiefly in the muscles of the shoulder, especially on the left side. The pains continued all day with great severity, sharp and lancinating in character and shooting through the whole length of the arms, although more severe in the shoulders. The next morning the pains had ceased entirely in the right arm, and the power of the extremity had returned. He was unable, however, to elevate his left arm and the severe pains still continued, especially in the region of the left shoulder. There were no vesical symptoms. For several nights following the onset of the paralysis he had chills of moderate severity. The patient was first seen by Dr. Hunt on Jan. 20, 1905, two weeks after the onset of the symptoms. He still complained of sharp, shooting pains in the region of the left shoulder, and the deltoid muscle was somewhat tender to deep pressure. It might be added, however, that on compressing the muscle bundles of the deltoid between the fingers the same pain and tenderness were elicited as on deep pressure. The nerve plexus above the clavicle and in the axilla and along the inner side of the arm was absolutely free from any tenderness. The left arm was the seat of a slight general atrophy, which was very marked in the deltoid muscle. All movements of the left arm and shoulder could be carried out with practically normal power, excepting adduction of the arm. On attempting adduction some of the posterior bundles of the deltoid were felt to contract, but little or no movement of the extremity was produced. All the arm-jerks were present, and were equal on the two sides. On testing the sensibility it was found that the tactile, pain and temperature senses were retained over both upper extremities, and that the deep sensibility was also normal. A more careful sensory test, however, showed that the sensation was relatively diminished for pain, temperature and touch along the outer side of the arm from the shoulder to the wrist, corresponding very closely to the recognized sensory distribution of the fifth cervical segment of the cord. On the right arm a similar relative obtunding of sensation was also present, but less marked and not so extensive, being practically limited to the region of the deltoid. The gross motor power and tendon reflexes of the lower extremities were normal. The Babinski phenomenon was absent. The deltoid muscle failed to respond at Erb's point to the galvanic current, although good contractions were produced in the biceps and the supinator longus. The direct faradic current produced a sluggish response in the posterior portion of the deltoid; no response in the anterior half. By the direct galvanic current a slow, vermicular response was elicited with reversal of the poles. The urine contained some albumin and hyaline casts. A blood count showed 4,112,000 red blood corpuscles, 10,000 white cells and 75 per cent. of hemoglobin. The erythrocytes were normal; no granular basophilia. The differential count was as follows: polymorphonuclears, 70.6 per cent.; lymphocytes, 10.2 per cent.; large mononuclears, 18.8 per cent.; eosinophiles, 0; mast cells, 0.4 per cent.

On Feb. 1, 1905, the patient still complained of a dull aching, with occasional sharp, shooting pains in the left shoulder. The moderate tenderness in this region still persisted. The gross motor power of the deltoid had greatly improved, so that the arm might now be elevated above the head. This occurred chiefly through the medium of the posterior half of the deltoid muscle, the anterior portion remaining entirely relaxed and flaccid. The slight sensory changes still persisted. There was no Babinski phenomenon. A small piece of dead bone had been removed from the inside of the mouth. The breath and excretions from the mouth were very fetid. The interesting features of this case, Dr. Hunt said, were those of localization and etiology. Considering the patient's previous good health and the absence of any other demonstrable cause, it was natural to refer the symptoms to the suppuration and the carious process in the lower jaw, more especially as mild symptoms of sepsis directly preceded and followed the onset. While the persistent pains and apparent tenderness over the left deltoid might suggest a neuritis of the circumflex nerve, it might be emphasized that the paralysis of the deltoid was incomplete, and evidences of objective sensory changes limited to the circumflex were absent. The speaker also emphasized the absence of tenderness or other evidences of inflammation in the plexuses and cellular structures of the neck. On the other hand, the initial paralysis and severe pains, symmetrical and bilateral in distribution, the rather sudden recovery of the right arm and the more gradual recovery of the left; the unequal distribution of the paralysis in the left deltoid, and the gradual improvement which this paralysis had undergone; the relative diminution of sensation on both sides in an area corresponding very closely to that of the fifth cervical segment, all pointed strongly to a central cord lesion. The nature of this case could only be inferred. A poliomyelitis or a myelitis of infectious origin naturally suggested themselves.

Dr. Charles L. Dana said he had seen Dr. Hunt's patient some days ago, when there was still some paralysis of the left arm. The condition could best be explained on the theory of a thrombosis, plugging one of the central arteries, and affecting the left anterior horn, about the region of the fifth cervical segment.

Dr. Fraenkel said there were a number of cases reported in literature where septic conditions, particularly that known as angina Ludovici —an acute suppurative condition of the connective tissues of the neck— had given rise to a septic neuritis. The speaker said he saw such a case in consultation about three years ago. The case was one of a septic throat condition, and eight or ten days later, after the acute symptoms had subsided, there were evidences of a bilateral brachial neuritis. It began with severe pain, in that way differing from the case shown by Dr. Hunt.

Address of the Retiring President, Dr. Pearce Bailey.—The speaker stated that while the membership of the Society had been materially enlarged during the past year, notably by younger men, the new members had not shown the active participation in the proceedings which was absolately essential for the Society's continued prosperity. Many interesting cases had been shown during the year, and the general discussions had brought out large audiences, and had been fruitful in valuable information. While individual clinical cases and general discussions perfected the art of the clinician, they did not give neurology all that neurology had a right to expect from as representative a body as the New York Neurological Society. For the science to be advanced, more fundamental contributions were essential. It was commonly said, and with much truth, that neurology was living to-day on its past achievements. So much the more necessary was it to try to give it new life, to start it again on that progress which, only a few years ago, made it the most brilliant branch of medicine.

Address of the President-elect, Dr. Joseph Fraenkel.—The speaker stated that while neurology had a glorious past, its future, viewed with the bias of the sentiment prevailing at present, appeared discouraging. A pessimistic attitude had crept in; the transactions of the meetings of neurologists seemed to lack the animation and enthusiasm of former days. Why this was so he did not know; it had happened because it had to—it was a phase of evolution. Dr. Fraenkel said that personally he did not believe that the present augured ill for the future. There was a wealth of fundamental questions awaiting investigation. New methods had been elaborated that promised valuable aid. Chemopathology, which seemed especially adapted for the elucidation of some neurological questions, with its promise of better therapeutics, was well established. The physiology of the ductless glands was closely allied to neurological themes and deserved particular attention. All neurologists were thoroughly dissatisfied with our conception and interpretation of the so-called functional conditions. It was encouraging to note a decidedly rational spirit in the newer literature on the subject. The relation of the nervous system to the pathology of the vegetative functions was a subject which the speaker believed to be of fundamental importance to clinical neurology and psychiatry. The subject had not been lacking in votaries, but had proven refractory and unyielding. A good deal of preparatory work had been done for the better understanding of the subject. So-called visceral or vegetative phenomena accompanied more or less prominently organic disease of the nervous system, and constituted the chief physiognomy of all functional states and functional symptom-complexes. Trophic, vasomotor and secretory disturbances of the skin, changes in the trophism of joints and viscera, disturbances of the physiologic rhythm of the vaso- and visceromotor innervation, perversions of visceral sensation, were often seen at the bedside, and were not sufficiently appreciated. The visceral and vegetative symptoms of tabes and hysteria were good illustrations of familiar types. The innervation of the ductless glands was manifestly a matter of importance, and the histology of the blood-vessels of the brain and their innervation were morphological questions of broad application to physiology and pathology. Phenomena of disordered trophic influence were not uncommonly encountered by the neurologists, and very little was known about most of them. The palmar and plantar *mal perforans* of tabes, the cutaneous ulcerations of syringomyelia, the decubital ulcers observed in the course of organic disease of the cord or brain, the hysterical skin eruptions, the glossy skin, scleroderma, morphea, etc., were hardly understood. Atrophy of the hair of the limbs in tabetics, and hypertrichiasis in cases of poliomyelitis were frequently seen. It was interesting to observe that in cases of disease of the anterior horns, when the disease was limited to the anterior horn cells, as in amyotrophic lateral sclerosis, for instance, this change in the growth of the hair did not take place. Atrophy of muscles severed from their connection with the peripheral nerves was frequently observed. Atrophy of visceral muscles under similar conditions was doubted. The trophic changes of the joints in tabes and syringomyelia, and the nature of arthritis deformans were still mysteries.' The pituitary body was generally believed to exert trophic influence upon some parts of the skeleton. A large number of vasomotor disturbances were still puzzling problems. The general vasomotor ataxias observed so frequently in the course of Graves' disease and the climacteric neuroses were, on clinical grounds, referred to perverted function of the thyroid glands or of the ovaries, respectively. Angioneurotic edema was believed by many to be explained on the basis of a toxemia. The question of erythromelalgia was still *sub judice*. Sachs and Wiener's findings of disease of the bloodvessels and peripheral nerves had been frequently corroborated. The condition was in danger of losing its clinical entity and of being classed with the arterial diseases of the extremities (angina pectoris of the foot, intermittent claudication). For explanation of the spasmodic disturbances of the respiratory function, asthma nervosum, tachypnea, hysteric dyspnea, little definite pathologic information was at hand. The bradycardia of direct and indirect vagal lesions was frequently described. According to Oppenheim, neuritis of the vagus was indicated by acceleration, rarely by slowing of the heart beat. The vast variety of symptoms referable to disordered innervation of motion, sensation and secretion of the gastro-intestinal tract were well known and often very perplexing. Cases of cord lesions were not infrequently met with which began with visceral, particularly gastro-intestinal symptoms, and which were often treated for a long time under this mistaken diagnosis. In a large number of cases of lead colic the celiac nerves and ganglia were found evidently diseased. Changes in the constitution of the urine were frequently observed in nervous diseases. Albuminuria was often a symptom of a pontine lesion, while glycosuria was frequently associated with disease of the posterior fossa. The symptomatology of disordered genital functions was also beginning to be studied along lines suggested by Dr. Fraenkel.

March 7, 1905.

The President, Dr. **Joseph Fraenkel**, M.D., in the Chair.

A Case for Diagnosis.—Dr. Smith Ely Jelliffe presented this case for diagnosis. The patient was a woman, 33 years old, a dressmaker by occupation. She had seven sisters and one brother alive and in good health. One sister had died of consumption. Her father, who was alive at 65, was a hemiplegic. Her mother was 70 years old, and enjoying good health. The patient had scarlet fever at the age of 7 years. Following this attack, she was weak for a long time and would fall easily. There was no history of hemiplegia at this time. Later in childhood she had measles, whooping cough and diphtheria. She had an attack of jaundice when she was 22 years old, and malaria six years ago. Her menstruation began when she was about 14, and was regular, lasting two days. Some years ago she had an attack of sciatica, and gave a history of painful swellings in the axillæ, elbows and knees, probably due to some lymphatic infection. When she was 18 she fell, striking the back of her head, resulting for a time in severe headaches.

Her present illness began two years ago, and its onset was gradual.

She first felt pain in the little finger of the left hand. This was burning in character, and ran up and into the shoulder along the course of the sterno-cleido-mastoid to behind the ear. The pains were persistent and grew more general. Then the hand began to shake and grow weaker. The pain had lasted two weeks before the tremor and weakness became apparent. The tremor at first was very pronounced. About a year later she noticed a loss of power in the left leg, with stiffness and drawing up of the muscles and some pain in the knee. During the past summer the pain had been referred to the ankle. She also complained of frequent attacks of diarrhea, with colic. There were no bladder symptoms. An examination of the eyes showed slight transitory diplopia, but no permanent nystagmus. No impairment of sight. There was slight transitory inequality of the pupils; no Argyll-Robertson pupil. Consensual light reflex normal. Skin reflexes normal, No pains; no anesthesia; no disturbance of heat and cold sensations; no Romberg. Slight tremor of head at times. Musculature good. There was weakness and stiffness of the entire left side, more marked in the upper than in the lower extremity. No sensory changes in the entire body. The reflexes were increased on the left side. No Babinski; no clonus; no tremor of leg. The tremor of the arm was coarse, of the semi-intentional type, but not typical. The heart sounds were exaggerated, but there was no murmur. The leg dragged; there was marked stiffness and rigidity. No electrical changes; no atrophy. The patient said that her memory was not as good as formerly, especially for recent events. Otherwise no change was noted. Her friends stated that she was more irritable than formerly, but this was perhaps natural, as the tremor of the hand prevented her from carrying on her business as a dressmaker. The speaker said the symptoms were suggestive of a multiple sclerosis, a spinal tumor, or a progressive hemiplegia of unknown causation.

Dr. L. Pierce Clark, who had also seen the patient at the Vanderbilt Clinic, said the case impressed him as one of multiple sclerosis, slowly progressive, and probably of the cerebral type. There were some evidences of involvement of the spinal cord.

A Case of Myotonia Congenita.—Presented by Dr. Graeme M. Hammond. The patient was a girl of 16, the youngest of three children. The other children were boys. One of them, aged 18, died suddenly a week ago of heart disease. The child's parents were cousins. There was no other case of myotonia on either side of the family for three generations; beyond that nothing was known of the ancestors.

The patient began to walk when she was ten months old. With the exception of measles in childhood, she had always been well and healthy. When she was about two years old the mother noticed that the child, after sitting awhile, had great difficulty in getting up and walking. She arose slowly, on account of great stiffness in the muscles, and often fell. On attempting to walk she at first found it impossible to move, so rigid were her legs. Finally she would move one leg stiffly, then the other, and began to walk with stiff, mechanical movements, like "a nursery doll." The more she walked the more limber she became, and in a few minutes walking became easy and natural, and she could even run and dance. This condition had persisted all her life.

She menstruated for the first time in March, 1904; then not again until September, 1904, and since that time she has not menstruated at all. Her body and arms were rather small, but the legs and thighs were very large. The knee-jerks, after sitting awhile, were present and responded quickly, but the movement was short. After walking, when the legs were quite limber, the knee-jerks were normal. Both calves measured 14¼ inches; the thighs (7 inches above the patella) 20¼ inches. The legs were 33 inches long. The muscles of the arm reacted normally to both forms of the electrical current. The legs seemed to respond normally to faradism. When galvanism was used the muscles responded sharply and deeply to make of current, then immediately relaxed, then a second contraction ensued, which was not so well marked as at first. The muscle, when it contracted, did so with a wave-like or vermicular movement. The same muscular tonus was observed after standing or lying down. The most difficult time the patient had was on attempting to rise in the morning.

Dr. G. L. Walton, of Boston, spoke of the liability of these cases to be mistaken for hysteria, on account of the apparently meaningless transition from clumsiness to agility. Continuous observation of the patients showed an unvarying adherence to fixed rules referable to disturbance of the neuro-muscular mechanism, and independent of the mind. Dr. Walton also referred to a symptom that was present in two typical cases of myotonia congenita that had been under his observation, namely, the slow and clumsy ascent of stairs, followed by an active and nimble descent. This trial was then made upon Dr. Hammond's patient, with the same result.

A Case of Progressive Muscular Atrophy of the Left Upper Extremities.—Presented by Dr. Edward D. Fisher. The patient was a young woman, whose occupation was that of a machine worker, passing articles along under a machine, the left hand being engaged more than the right. Her family history was negative as was also her past history, with the exception of the fact that when she was about ten years old she complained of pain in the upper arms, between the shoulder and elbow, when carrying her school books.

About two years ago she began to notice some weakness in the left hand, together with some tendency to stiffness, especially when playing the piano. This was followed by wasting of the muscles, which appeared to be typical of progressive muscular atrophy. One peculiarity of the case was the extreme icy coldness of the hands, in spite of efforts to keep warm. The electrical reactions were impaired. There were no sensory disturbances; no symptoms pointing to syringomyelia; no scoliosis. The atrophy was gradually extending up the forearm.

Dr. William M. Leszynsky said the case reminded him of one he saw recently in which there were various symptoms affecting the upper extremity, and in which the presence of a cervical rib was subsequently demonstrated. This abnormality, the speaker thought, should always be looked for with the X-ray in doubtful cases of this character, and wauld, perhaps, often explain symptoms which would otherwise be attributed to some lesion of the brachial plexus.

Dr. L. Pierce Clark said that he had recently seen two cases of early progressive muscular atrophy in which the symptom observed in Dr. Fisher's case, that of coldness of the skin, was present. However, the symptom was confined quite distinctly to the skin distribution of the ulnar nerve in one case, and in the other the coldness occurred in the entire skin area of the first dorsal segment of the cord. He believed, in these cases at least, that progressive muscular atrophy was a lesion of the peripheral nerves as well as of the anterior horn of the cord, consequently the sympathetic nerves were damaged, as in traumatic peripheral neuritis. Recent autopsy studies on progressive muscular atrophy would seem to confirm this pathological explanation.

Dr. William G. Spiller of Philadelphia said that in his experience progressive muscular atrophy of spinal origin was one of the rarest of all nervous diseases. He recalled one case, reported by Dr. C. S. Potts from the University of Pennsylvania's nervous clinic, in which the atrophy began in the extensors of the forearm. The explanation of the condition in that case was that the man's occupation was that of a fireman, and the atrophy began in the muscles that were most constantly used.

Two Cases of Nerve Anastomosis.—Presented by Dr. Alfred S. Taylor. The first patient was a boy, 8 years old, who three years previously developed an attack of anterior poliomyelitis which involved the right hand and forearm. The hand was entirely useless, but as the muscles of the shoulder and upper arm were in fairly good condition, the case was regarded as a suitable one for nerve anastomosis.. Accordingly, a few days ago the brachial plexus was exposed and the 7th and 8th cervical and 1st dorsal roots were divided, turned up and inserted by lateral anastomosis into the junction of the 5th and 6th nerves. The anastomosis was effected without any tension on the nerves. The wound was then closed and the parts immobilized. The dressings were still in place and the ultimate outcome of the operation, so far as improvement of function was concerned, was doubtful.

The second patient, shown by Dr. Taylor, was a boy, about 8 years old, who in the early summer of 1903 had scarlet fever, followed by otitis media and mastoiditis, for which he was operated on July 13, 1903. at the Randall's Island Hospital. Three days later a facial palsy appeared, and rapidly progressed to complete paralysis. On October 24. 1903, three and one-half months later, complete R. D. was found on electrical examination. A facio-hypoglossal anastomosis was immdeiately done under chloroform anesthesia. The facial nerve was found to be soft and friable, and gray in appearance. But one suture could be made to hold to the hypoglossal, which in turn was under such tension that a fine suture was passed through the hypoglossal sheath to anchor the nerve leading to the neighboring fascia, and so prevent tension on one anastomosis, as far as possible. Primary union was obtained. There was almost complete palsy of the muscles supplied by the hypoglossal immediately after the operation. Ten weeks after the operation some difficulty of speech remained, owing to imperfect use of the tongue. Difficulty in swallowing and mastication, and unilateral furring of the tongue, all of which were present for some weeks after the operation, and which were due to operative injury of the 12th nerve, disappeared. Three months after the operation the face was far less assymetrical than before. and the eye could be partly closed. R. D. was unchanged. At the end of eight months there was slight motion about the right side of the mouth. The improvement slowly continued and now, fourteen months after the operation, the patient had a fair amount of control over the formerly paralyzed side of the face.

Dr. B. Sachs thought that three and one-half months was rather too soon to consider an operation in a case of facial palsy, as in many of these patients improvement began after a much longer period than that which had elapsed.

Dr. Hammond said that he did not think the return of power in the paralyzed muscles in the case shown by Dr. Taylor proved that the anastomosis had been successful. He recalled the case of a young lad who developed a facial palsy after a mastoid operation, and without any attempt at anastomosis, motion in the affected muscles was first noticed at the end of fifteen months, and eventually was absolutely restored.

Dr. Leszynsky said he had seen a great many cases of Bell's palsy following mastoid operations, and in his experience a favorable outcome, like that reported by Dr. Hammond, was rather unusual. He had followed some of his cases for a long time, and had failed to note any improvement.

Further Report of a case of Hypoglossal Facial Anastomosis.—By Dr. Charles H. Frazier, of Philadelphia. The speaker said that his first opportunity to operate upon a case of facial palsy was in August, 1903. when he saw, in consultation with Dr. William G. Spiller, a patient who, five months before, in a fit of mental aberration had shot himself with a revolver. the ball entering the external auditory meatus. This resulted in a complete facial palsy, and from the manner in which the wound was inflicted and the point of entrance of the bullet, there was every reason to believe that the facial nerve was completely severed in its passage through the petrous portion of the temporal bone before it made its exit from the stylomastoid foramen. The case was one eminently suitable for operative intervention, as the nerve was irreparably damaged and the muscles hopelessly paralyzed. The operation consisted in a hypoglossal facial anastomosis. During the eighteen months that had elapsed since the operation, which was done in September, 1903, the patient had been examined from time to time, and there had been a slow, but progressive improvement.

One month after the operation there was no response to the faradic current in any of the muscles. Reaction of degeneration and polar changes were marked. Six months after the operation there was no change in the condition. Nine months after the operation there was still no response to the faradic current, while with the galvanic current there was lessened irritability. At this date there appeared for the first time a peculiar involuntary movement of the angle of the mouth. Fourteen months after the operation there was still no response to the faradic current, but a slight increase of irritability under the galvanic current. Polar change still distinct. Seventeen months after the operation voluntary movements at the angle of the mouth were for the first time observed. In addition to this the patient was able to almost completely close the eye. All the muscles responded to the faradic current.

Dr. Frazier called attention to the fact that it was not until after the fourteenth month had passed that a positive reaction to faradic stimulation was observed, with one exception, namely, the orbicularis palpebrarum, which responded slightly in the ninth month. Another interesting point was the restoration of an involuntary before a voluntary movement. In those cases in which the anastomosis had been made with a branch of the trunk of the spinal accessory nerve, associated involuntary movements were the rule rather than the exception. The speaker called attention to the order in which the muscles regained their power, beginning with the muscles of the lower part of the face, those supplied by the branches of the cervico-facial division, and extending gradually to the upper part of the face, or those supplied by the tempero-facial division.

One of the arguments that had been advanced against the selection of the hypoglossal nerve was the resulting atrophy of the tongue. Section of the hypoglossal nerve would be followed by paralysis of the depressors and some of the elevators of the hyoid bone, with atrophy of one-half the tongue. The speaker said that apart from the misshapen appearance of the tongue, which was not to be compared in importance with the facial deformity one was striving to relieve, he had not found any other disturbance arising from section of the hypoglossal nerve. In none of his cases was there difficulty in swallowing or talking.

Dr. Frazier said he thought no one would question the propriety of the operative treatment of facial palsy. The operation was founded upon a psychological law, proven by experimentation, and the clinical experience of a number of observers had established all that was claimed for it. The only feature in the technique of the operation worthy of discussion was the choice between the eleventh and twelfth nerves. In a previous communication on the subject (University of Pennsylvania Medical Bulletin, November, 1903) the speaker said he expressed a preference for the twelfth nerve, and from subsequent investigation of the results of other operators and from his own experience, he still held to the same opinion. The whole question hinged upon the importance which was attached to the associated movements. So far as he had been able to learn from the recorded cases of spinal-accessory-facial anastomosis, there had not been a single instance in which a voluntary effort to raise the shoulder had not been attended with contraction of the facial muscles, and these so-called associated movements constituted a very serious objection to the selection of the eleventh nerve. As to the method of making the anastomosis, theoretically, an end-to-end anastomosis should secure the best results.

Some Newer Ideas on Nerve Anastomosis.—By Drs. William G. Spiller and Charles H. Frazier.

A Contribution to the Pathology and Surgical Treatment of Chronic Facial Palsy. By Drs. L. Pierce Clark, Alfred S. Taylor and Thomas P. Prout. (Read by Dr. Clark.) The reader said it was not known why the facial nerve suffered from palsy with such frequency, nor was there as yet adequate information regarding the nerve elements or the part of the nerve that was primarily involved. The tendency of Bell's palsy was probably due to anatomic causes. The nerve had a tortuous and exposed course through the Fallopian canal, and the extension of inflammation from the middle ear, or the effect of cold acting through this channel upon the facial nerve, was easily understood. Facial palsy, both in man and animals, was relatively infrequent when the face and head were properly protected. It seemed reasonable to suppose, also, that a certain number of individuals suffered from a congenital narrowing of the foraminal exit of the nerve.

There were good reasons to believe that the initial changes of this condition took place in the perineural sheath, although the exact pathogenesis of Bell's palsy was still uncertain, because of the meagerness of the material at hand for study. In two cases of typical, uncomplicated Bell's palsy coming under the author's observation and treated by facio-hypoglossal anastomosis, a section of the affected nerve was excised and submitted to Dr. Prout for microscopical study. In the first of these cases the paralysis had existed for three and one-half months; in the second, for twelve years. Dr. Prout reported that in the first case the nerve was in a condition of incomplete degeneration. There were still a few nerve fibers undegenerated, or in which the myelin sheath was only partially broken up, and these stood out in marked contrast to the degenerated nerve fibers in which they were imbedded, which latter showed here and there fragments of myelin. There was, furthermore, a marked invasion of the tissues with leucocytes, and the cells of the neurilemma were shrunken and shrivelled in some portions; in others the nuclei of the neurilemma cells were large and well-defined. The perineural sheath showed marked infiltration with leucocytes.

In the second case, which was of a chronic character, the nerve fibers were completely degenerated, showing but occasional granules belonging to the myelin sheath. There were no undegenerated fibers. The tissues belonging to the perineural sheath showed the most decided changes. There was marked proliferation of the cells belonging to the perineural sheath, with infiltration of the tissues, and clumps of cells scattered here and there throughout the section. This tissue exceeded the proper nerve tissue of the section by about two to one. The neurilemma cells were markedly shrunken and shrivelled.

The painlessness of Bell's palsy even to external pressure on the nerve trunk and the paralyzed muscles argued for a simple degenerative neuritis in the peripheral portion, and not a parenchymatous affection. There was good reason to believe that Bell's palsy was primarily a Fallopian neuritis, which through strangulation of the nerve strands induced a low grade of secondary degenerative neuritis in the periphery.

Dr. Alfred S. Taylor said that his technique of facio-hypoglossal nerve anastomosis was described in detail in a paper upon the surgical treatment of facial palsy by Dr. L. Pierce Clark and himself, which appeared in the *N. Y. Medical Record*, February 27, 1904. The operation involved the following steps: (1) The incision. (2) Isolation and section of the facial nerve. (3) Exposure of the hypoglossal nerve. (4) The implantation. (5) The closure of the wound. (5) The after-treatment.

The incision, involving the skin and subcutaneous tissue, passed along the anterior margin of the mastoid process and the sterno-mastoid muscle for about 5 cm., starting at the level of the external meatus. When the facial nerve was identified, it was enucleated from the surrounding connective tissue, and divided as far up in the stylo-mastoid forearm as a narrow-bladed knife would allow. Usually one could get from one to two cm. of free nerve trunk.

The isolation of the hypoglossal nerve was the most difficult and tedious step, and involved whatever danger there was in the operation. Once identified, it was dissected upward until the stump of the facial nerve could be approximated to it without tension. This had to be done with care, so as not to divide the minute branches from the pneumogastric, the upper ganglion of the sympathetic and the two upper cervical branches, all of which were in the immediate neighborhood. While the nerve was supported on a blunt hook, a slit one-half cm. long was made well into the nerve trunk. A fine curved needle was threaded to one of each pair of long silk ends previously left tied to the stump of the facial nerve. One suture was passed through the inner and the other through the outer margin of the wound in the hypoglossal nerve. When the sutures were tied the wedge-shaped end of the facial was snugly held in the cleft of the hypoglossal nerve, and was usually best turned slightly upward by means of a probe, a procedure suggested by Dr. Weir. These sutures should not be tied too tightly, lest they injure the fibers of the hypoglossal nerve, a few of which were almost surely included in their grasp.

To prevent the ingrowth of connective tissue elements, Cargile membrane was wrapped about the nerve junction.. The hypoglossal was then dropped back to its normal position, and there was usually no tension on the tissues.

The after-treatment was a most important feature in obtaining the desired result. Massage, electricity, and later coördinate muscular movements, should be persistently and systematically resorted to for months.

As to the time when the operation should be done, Dr. Taylor said that that must be left to the neurologist. The muscles should be kept in good condition. It was known that there was a return of motor power in some instances even after many years had elapsed.

In connection with his remarks Dr. Taylor reported six cases of hypoglossal nerve anastomosis, and showed a number of new instruments that he had designed for the purpose of rendering the operation easier technically, and of avoiding distressing sequelæ. These instruments were illustrated and described in detail by Dr. Taylor in the *N. Y. Medical Record*, March 4, 1905, page 359.

Dr. Charles A. Elsberg reported the following case of facial-spinal-accessory anastomosis. This report, he said, must be regarded as preliminary, as the time that had elapsed since the operation was not long enough for a complete report.

C. F., female, 30 years old, was referred to Dr. Elsberg in May, 1904, by Dr. H. Heiman. When the patient was six months old she had an attack of convulsions, followed by paralysis of the left side of the face, with loss of smell and hearing on the same side. In spite of treatment, the paralysis did not improve. Massage, electricity, injections of strychnine, etc., were continued for many years without any improvement.

When Dr. Elsberg first saw the patient she presented the characteristic features of an old facial palsy. Dr. B. Sachs was asked to see the case in consultation, and found that the left facial paralysis was almost complete. There was a slight faradic response to the lowermost branch of the nerve and the galvanic response was diminished, but the polar formula was not altered in the orbicularis oris or in the nerve itself. There was partial lagophthalmus. Examination of the ear and nose was negative.

After careful consideration it was decided that, in spite of the long duration of the paralysis, an operation might be attended with success. This was done on May 10, 1904, about nine and a half months ago. The facial nerve was anastomosed with the spinal accessory by slitting both nerves and joining the branch of the 11th nerve to the trapezius muscle to the separated portion of the facial with fine silk sutures. Thus neither the 11th nor 12th nerves were entirely divided. The wound healed by primary union, and the patient was discharged from the hospital in two weeks.

Briefly, the after-course was as follows: There was complete paralysis of the trapezius and a partial paralysis of the left sterno-mastoid after the operation. This disappeared in about six months. Eighteen days after the operation the patient declared she had a continual feeling of tenseness in the left side of the face. On the 26th day she declared that she could close her eyes better, and that the left eye did not tear any more. On the 96th day the patient wrote from the country that she had noticed that she was able to move the left corner of her mouth a little, and that she could close the left eye better.

On the 122d day the orbicularisoris contracted strongly to the faradic current; the muscles of the lower lip slightly to galvanism. The same muscles contracted when the spinal accessory was stimulated. Associated movements of the left shoulder and the left side of the face could never be obtained. On the 166th day the patient was able to draw the left angle of her mouth slightly upward and outward, and could close her left eye almost completely. From that time up to the present there had been a slow, but steady, improvement, additional muscles beginning to contract to galvanism and faradism every few weeks.

The case was unusual on account of the duration of the paralysis at the time of operation (29½ years), and because of the absence of associated shoulder movements.

Dr. B. Sachs said that in deciding the question whether to anastomose the facial nerve with the hypoglossal or the spinal accessory his preference would be in favor of the latter procedure, as it involved a nerve of lesser dignity than the former. The hypoglossal was a nerve of great importance, and if the same result could be obtained by substituting the spinal accessory he would certainly favor it. The entire question of nerve transplantation was an extremely important one, and the physiological aspects of the problem were still unsolved. The speaker said he sincerely hoped that the procedure would prove beneficial in hemiplegia, but until further experimental work had been done that aspect of the subject could not be intelligently discussed. In facial palsy several cases had been reported where the nerve anastomosis had been done three and a half or four months after the onset of the disability. This, he thought, was too early, as many cases were on record where improvement began much later without treatment or in spite of treatment. A year at least should be allowed to elapse before operation.

Dr. Leszynsky said he was interested in the remark made by Dr. Frazier that the power would be restored with the return of the faradic irritability. The speaker said he had seen patients with facial paralysis of long standing in whom the faradic irritability had returned although there was no return of motility. These were exceptional cases, but they had been observed, so that the mere return of the faradic irritability did not necessarily indicate that there would be a return of motility. The speaker said he agreed with Dr. Sachs that three or four months was too soon to operate on these cases.

Dr. Walton said that Dr. Spiller's proposition should have the benefit of trial. In disease or injury of the lower neurones every gain in motion, however slight, was gladly welcomed, and if hemiplegics could be helped by nerve transplantation, there was the added hope of lessening the cerebral spasm to which Dr. Spiller had referred, and which in one case coming under the speaker's observation was so troublesome that the arm was amputated at the shoulder at the request of the patient. The brilliant result in Dr. Elsberg's case demonstrated the possibility of transplanting the spinal accessory without causing associated movements, which seemed to be the chief drawback to shoosing this nerve in preference to the hypoglossal.

Dr. Spiller, in closing, said the statement made by Dr. Taylor that in late cases, where the paralysis had existed for a long time, the nerve may have disappeared, was correct. The speaker recalled such a case where the facial nerve could not be found at all. In facial palsy one of the earliest evidences of a return of power that could be elicited was a slight deepening of the naso-labial fold when the eyes were closed tightly.

Dr. Frazier, in closing, said that in his cases of nerve anastomosis the hypoglossal had been selected in preference to the spinal accessory purely on theoretical grounds and a study of former operations, and also because associated movements had followed the selection of the spinal accessory. The case reported by Dr. Elsberg was the first one in which these associated movements had not occurred. Henceforth, Dr. Frazier said, he would favor the end-to-side amastomosis in preference to the end-to-end method.

Dr. Clark, in closing, said he thought the proper time to operate could be fairly accurately decided upon by the results of the electrical tests, and he did not think it wise to fix an arbitrary period of time. In most forms of peripheral neuritis the electrical tests formed a basis of prognosis that was almost absolute. Certainly, at the end of three or four months, if the electrical changes should complete degeneration of the nerve and there was no evidence of repair, he thought an operation would be indicated. At the same time, if one preferred to wait a year, or even thirty years, he saw no objection to it.

Dr. Taylor said the disappearance of tearing to which Dr. Elsberg referred in his case had also been noted in one of his cases. The operative damage to the hypoglossal and the tongue symptoms resulting therefrom were only temporary, and he did not think they should be looked upon as a serious objection to the selection of that nerve for the anastomosis.

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 28, 1905.

The President, Dr. Joseph Sailer, in the Chair.

A Case with Some of the Symptoms of Multiple Sclerosis Due to Trauma.—This paper was presented by Dr. Ralph Pemberton.. On the 16th of January, 1905, the man fell from the upper girders of the Subway, where he was working, a distance of about 16 feet, though whether he stepped into some opening or lost his balance while at work he cannot say. He became unconscious and does not know how he struck, though he fell on a cement flooring. He was taken to the Medico-Chirurgical Hospital and was treated there for four weeks, of which time he can give no satisfactory account, though he was not constantly in bed, and was told that at times he had to be catheterized. On two occasions plaster jackets were applied, the second of which alone he remembers. He does not know how long he was unconscious, and since the fall he has had constant abdominal tenderness, pain in the back and constipation, and he states that before the fall he had been feeling well except for a bad cold.

He has never been subject to fits or periods of unconsciousness as far as he knows, and has always been of an active habit.

Examination.—He is a well-built and symmetrically developed young man. No paralysis of facial or ocular muscles is present, pupillary reactions are normal and there is no nystagmus. The tongue protrudes promptly in mid-line, and is very slightly tremulous. There is at times an antero-posterior tremor of the head, especially when he is standing and therefore supporting the head, but also when in bed and when a voluntary act, as touching his fingers to his nose, is attempted. His mentality seems fair and he answers questions well, but his speech is slightly tremulous, and a relative states that it was not so formerly. Chest and intra-thoracic organs and abdomen seem normal, except that over most of the abdomen is a diffuse light-brown discoloration extending over the genitalia, which the patient says is congenital.

When first admitted to the hospital patient complained of considerable abdominal pain and tenderness to palpation, most of which disappeared by the next day. The spinal column is straight and presents no abnormality to inspection, but in the lower thoracic region, extending down to about the sacrum, pressure over the spines of the vertebræ causes pain. Pain is also elicited by pressure over the region of the left tuber ischii.

The patient lies quietly in bed and can move both arms freely, though a coarse tremor is evident on both sides, especially the right, when any voluntary act is attempted, and is most marked in the finger to nose movement. This he performs rather slowly, uncertainly and waveringly even with his eyes open, but better with the left hand. Occasionally when the limb is quiet the tremor is present on the right side, but on extending both hands the fingers do not seem to be involved. Muscular power in the hands and arms is fair and equal, though hardly commensurate with his general musculature. The biceps tendon reflexes seem about equal and very slightly, if at all, exaggerated on both sides, and the triceps reflexes are about normal.

The legs lie quietly and can be raised freely, move at all joints and show a fair degree of power, though he complains that flexion of thighs on abdomen causes pain in the groins.

The patellar reflexes are prompter than normal and somewhat exaggerated; there is no patellar clonus, though a tendency to it is sometimes apparent, and no ankle clonus is present. Plantar stimulation on both sides gives some dorsal flexion of all the toes, and occasionally of the great toes alone, though the movement is hardly typical. The Achilles jerks are normally present, and tapping the patellar tendons at times throws the legs into spasm. On raising either leg voluntarily a slight intention tremor is noticeable. Sensation in all parts is apparently good. The arms are of fair musculature and size, but the legs seem small, and the calves especially are soft and flabby.

He moves slowly, gets out of bed cautiously, and walks slowly and carefully, with his back slightly bent forward, but his station is good with eyes closed or open. Walking or standing up straight causes pain in the groins, according to his statement, but when quiet in bed he is comfortable. Ophthalmoscopic examination by Dr. Oliver on March 21, 1905, for pallor of the optic discs showed the eye-grounds healthy in every detail. Since admission to the hospital two weeks ago he has improved somewhat; the intention tremor is less pronounced, and he says he feels better.

Dr. Dercum referred to a case he recorded some years ago of a man who fell from South Street bridge and afterward died. This man had tremor as a marked symptom, but there was no change whatever in the central nervous system suggesting spinal sclerosis.

Dr. Gordon thought the intention tremor of the right hand to be the only symptom he could see of disseminated sclerosis. He called attention to the continuous passive tremor of the head. In a case which had come under his observation there was first a tremor of the head similar to this case, and it developed later as paralysis agitans, and he thought it possible that this case of Dr. Pemberton might be one of paralysis agitans.

Dr. Lloyd said this case reminded him much of the cases of hysterical tremor described by French authors. Luys once reported a case which he claimed was one of paralysis agitans, which he had cured under hypnotism with rotating mirrors. Rendu and others took exception to Luy's diagnosis, and this led Rendu to write his classical paper on hysterical tremor. Westphal also reported two cases of so-called insular sclerosis in which the autopsy revealed no lesions. In a former paper on hysterical tremor Dr. Lloyd reported a similar case. Some years ago such a case was seen at Blockley in a man who had experienced a severe earthquake shock in South America. His condition and tremor were very similar, although worse, to that of Dr. Pemberton's case, and had lasted for several years. But he eventually entirely recovered. Dr. Lloyd believed that such cases are instances of traumatic neurosis, or hysterical tremor.

Rendu pointed out the characteristics of this tremor. It is sometimes wide in range (although not always), and it is much exaggerated by voluntary motion, just as in insular sclerosis. It differs, however, from insular sclerosis in the fact that a tremor is usually felt when the patient is at rest, as by placing the hand lightly on the patient's head. This is so in Dr. Pemberton's case. Dr. Lloyd was inclined to believe that the case was one of traumatic hysterical tremor, and that the patient could be cured by suggesive therapeutics.

Dr. Spiller called attention to the fact that Dr. Pemberton's case was not reported as one of multiple sclerosis, but simply as a case with some of the symptoms of multiple sclerosis, in order to show how difficult it may be to diagnose between hysterical tremor, organic injuries of the central nervous system and multiple sclerosis.

Dr. Pemberton read a paper on the direction of turning in organic hemiplegia.

A Case of alcoholic multiple neuritis with exaggeration of the knee-jerks and a paper on the regeneration of peripheral nerves were presented by Dr. S. D. Ludlum.

Dr. J. T. Krall and Dr. T H. Weisenburg exhibited a case of injury to the head with involvement of some of the cranial nerves.

Dr. Charles K. Mills exhibited a case of choreo-athetoid movements of one upper limb.

Dr. Eshner though this case would recall to many members of the society the classical case of Dr. S. Weir Mitchell, in which the patient presented a violent tremor of somewhat varying character, which ceased during sleep and could be checked by lying down. For a long time Dr. Mitchell thought the condition an anomalous form of spasm or tremor of obscure origin, but of recent years he decided that the case was hysterical. Under hypnotic suggestion the man was entirely relieved. The symptoms returned, however, under some emotional strain, and they then continued until death, which occurred suddenly, and on post-mortem examination was found to be due to heart disease. No changes in the nervous system were found.

Dr. Dercum remembered the case Dr. Eshner cited, and stated that the man had no other stigmata of hysteria except the tremor. He thought this case and the one Dr. Mills exhibited very similar.

Dr. William Pickett exhibited a case with difficulty in urination.

Dr. Mills thought the case might be one of sacral tabes. He considered that the loss of the Achilles jerk was suggestive of that condition. He recalled a paper he had read several years ago on the subject of the Achilles jerk and other reflexes in which loss of the Achilles jerk was the earliest and almost the only symptom present in two cases which afterward developed other tabetic symptoms.

Dr. Gordon believed the case to be one of tabes for the following reasons: Loss of Achilles tendon reflex has been observed by many writers in the preataxic period. The knee-jerks are also diminished on both sides. The occasional pains in the legs and the condition of the pupils, also some disturbance of the function of the bladder—all these symptoms are certainly in favor of tabes. Although all the symptoms outside of the tendon Achilles reflex are not pronounced, they are nevertheless grouped together to add to the value of the complete absence of the Achilles tendon reflex.

Dr. Robert H. Chase read a paper entitled Insane Delusions.

Dr. Dercum said we must all admit the great rôle which the feelings play in the development of delusions. He had approached the subject of mental diseases rather from the standpoint of internal medicine than from the standpoint of psychology.

Dr. Lloyd said that Dr. Chase had given a very careful analysis and classification of delusions. While delusions may thus be classified with advantage for purposes of study, it is, of course, not possible to classify the various forms of insanity merely according to the delusions; nor, as he understood him, does Dr. Chase intend to do this.

Some of the points touched on by Dr. Chase are very profound ones. Dr. Lloyd thought one of the most interesting analyses of the distinction between emotion and intelligence has been made by Ribot in his work on the emotions, and this pursues in great detail some of the very ideas which Dr. Chase has embodied in his paper. Ribot's idea is that in the development of the mind, emotions precede the intellect; and he likens the very earliest impulses of organic life in the vegetable cell—such as the impulse to take and assimilate food—to the beginning of the emotions or impulses in the primitive animal cell. It is doubtless true that these very fundamental processes or feelings are involved in the formation of delusions. The sense of personality is altered in various ways.

Dr. Lloyd had sometimes thought that a good test would be that a man is never insane until his ego is insane. A man may have very extravagant opinions on all sorts of subjects, such as religion, politics, or every-day affairs, but he is not necessarily insane on that account. This is a matter of common observation. We all know what incredible opinions are entertained on religious subjects by wise and educated men. But these opinions are, as it were, outside the domain of the ego. They do not affect the personality of the believer. The Moslem who believed that Mahommed went to the seventh heaven and returned in an instant of time is not insane, although his belief is extravagant. But it does not involve his own ego. If, however, a man believes he is a Messiah, it is a different story. Such a man is insane. So it is with the persecutory lunatic; the whole delusional cycle centers upon the ego. It was Griesinger who also elaborated this idea of the alteration of the personality, of the sense of the ego, in insane delusions.

Dr. C. L. Leonard read a paper on the treatment of some neuralgias by the Roentgen rays.

CHICAGO NEUROLOGICAL SOCIETY, IN JOINT MEETING WITH THE CHICAGO MEDICAL SOCIETY.

March 29, 1905.

The President of the Chicago Neurological Society, DR. HAROLD N. MOYER, in the Chair.

Symposium on Exophthalmic Goitre.—Dr. L. F. Barker opened the discussion on the symposium with a paper on:

The General Considerations.—He said the disease had been named after investigators in several countries, but the syndrome was undoubtedly very well described by Parry earlier than by any of the authors mentioned, and strictly speaking, we should, therefore, if we refer to the disease under the name of any one man, call it Parry's disease.

Four principal theories of the disorder have been put forward: (1) That it is due to disease of the sympathetic nervous system; (2) that the seat of the malady is in the medulla oblongata; (3) that it is primarily a disease of the thyroid gland, and (4) that it is a neurosis.

Modern therapeutic measures have been largely based upon the thyroid theory, and the results of therapeusis have been adduced in support of it. There is no gainsaying the fact that the results of partial strumectomy indicate that the successful removal of a portion of the thyroid gland can lead to pure or to definite amelioration of the condition.

The theory that exophthalmic goitre is primarily a neurosis, first championed by Charcot and his pupils, still has supporters among the ablest men of the profession. A survey of the whole subject makes the speaker personally unwilling for the present to deny it.

As to treatment, experience has shown the great importance of general measures; complete rest for a time, fresh air, careful diet, mild balneotherapy, all are useful. Dr. Barker personally favors rest, isolation and systematic psychotherapy as a routine treatment. In cases which do not respond to this or to antithyroid therapy operation may be considered, but if it is to be done it should be undertaken before marked cachexia develops, and before the heart is too seriously diseased. Before any safe conclusions can be drawn regarding any form of treatment a large number of cases should be observed over long periods. Results so favorable as to be startling have been obtained from the most diverse forms of treatment, even from feeding thyroid as well as from antithyroid medication.

The Larvated Forms of Exophthalmic Goitre.—Dr. L. Harrison Mettler, in his paper on this subject, stated that there are those who find exophthalmic goitre to be a rare affection, while others regard it as common. A tabulation of its so-called symptoms shows upwards of 75 clinical phenomena. In order to get at something definite, the author separated these clinical phenomena into (a) typical symptoms; (b) doubtful symptoms; (c) mere complications and associations. The atypical cases are much more common than are usually supposed, enjoy a comparatively favorable prognosis, and are highly amenable to treatment.

There is a large class of cases, the *formes frustes,* so much studied by the French authors, in which tachycardia with general nervousness, tremor and possibly slight evanescent struma or exophthalmos occurs. The heart irregularity comes on suddenly after a slight strain or sudden shock, mostly in a woman with hysterical tendencies or with a neuropathic ancestry. It exhibits the typical traits of the Basedow tachycardia, such as the persistent elevation of the pulse beat, the peculiar thrill. With it is a general nervousness and possibly a number of symptoms, such as a mild psychosis, hyperidrosis, vertigo, aboulia, weakness, and even some general emaciation. These cases are not infrequently mistaken for simple neurasthenia, tuberculosis and other affections.

Ocular Symptoms of Graves' Disease.—Dr. Henry Gradle discussed this phase of the subject and remarked that the exophthalmos may be so pronounced that the lids cannot be closed. Even partial forward dislocation of the eyeball has been seen on attempting forcible closure of the lids. On the other hand, the exophthalmos may be inconspicuous or not alike in the two eyes. One-sided exophthalmos is not excessively rare.

The retraction of the upper lid exposing the sclera to an abnormal extent it is perhaps more characteristic of Graves' disease than mere protrusion of the eye. It is less often absent than the exophthalmos, and, like the latter, it is occasionally, though rarely, one-sided. In rare instances it is present without exophthalmos.

Pigmentation of the skin, resembling the bronze color of Addison's disease, has been noted especially by Drummond on various areas of the body and also around the eyes. Jellineck has lately again referred to the diffuse brown coloring of the skin of the eyelids, the upper more than the lower, as an early, though not constant and sometimes evanescent, symptom in exophthalmic goitre.

Pulsation of the arteries of the retina has been described by Becker as common in this disease. Other observers have not seen this pulsation as often as Becker.

The protrusion of the eyeball is partly due to increased vascularity of the orbit, as it always diminishes after death. In some autopsies the eyeballs, prominent in life, were even found receded to normal position, and no anomalies were observed in the orbit. In other post-mortem records the exophthalmus was explained by the increased amount of fat found within the orbit. The ocular muscles were infiltrated with fat and the optic nerves elongated.

When recovery occurs in this disease, either spontaneous or under treatment, the eye symptoms can disappear completely. Some instances have been observed, however, in which a slight degree of exophthalmos existed after apparent recovery from other manifestations of the disease. If the disease continues without improvement, there is no reason to fear any eye complications or sequels except danger to the cornea in relatively rare instances. When the gaping lids afford insufficient protection to the exposed cornea, ulceration of the latter may take place. This does not seem to be a frequent occurrence, especially in recent times.

The Serum Treatment of Exophthalmic Goitre.—Dr. Harold N. Moyer said that since Moebius, Ballet and Enriquez and other investigators conceded that exophthalmic goitre was due to a fundamental derangement (an excessive or perverted secretion of the thyroid gland, considerable progress has been made in the treatment of this affection. If the disease be the result of a too abundant or an excessively toxic secretion of the thyroid gland, the only rational form of therapy consists either in checking this secretion or else in neutralizing the toxic products thrown into the blood stream. The first of these alternatives was beyond our control, but, as the thyroid secretion is perhaps normally neutralized by certain anti-bodies present in the blood, the serum or blood of animals from which the thyroid gland had been removed and in which these anti-bodies had been allowed to accumulate for some time should prove capable of neutralizing the excessive or perverted secretions causing Graves' disease.

The first experiments conducted along these lines were undertaken independently of each other by Ballet and Enriquez in Paris, and Lanz in Amsterdam. The former used the serum, the latter the milk of thyroidectomized animals. The results obtained by either method were very encouraging. Further experiments along these lines were conducted by

Burghart, Schultes, Goebel, Moebius and others, some using the milk, others the blood of thyroidectomized animals. The same properties which were found in the blood and serum proved to be present also, although in not as great a concentration in the milk of these animals. The latter was preferred by some experimenters, because it proved less expensive than the pure sera. The serum was at first administered subcutaneously, but as it was found that the same results could be obtained by using slightly larges doses *per os,* this latter method was considered more desirable.

H. Hempel and K. Thienger both report favorably upon the use of Moebius' anti-thyroid in serum, as prepared by Merck & Co. The harmlessness of all these preparations is self-evident, and all reports unanimously attribute to them much therapeutic value in cases of exophthalmic goitre. The new preparation, thyroidectin, is derived from the blood of thyroidectomized animals. In the preparation of these capsules not only the serum, but the entire blood, has been utilized. It forms a reddish-brown powder, readily soluble, non-toxic, unirritating to and rapidly absorbed from the stomach. It is put up in five-grain capsules, of which one or two are administered three times a day, according to the needs of the individual cases. This remedy has fairly passed the experimental stage; it has been placed in the hands of prominent neurologists in this country, who are careful observers and have testified to the fact that it possesses at least the greatest palliative value in the treatment of exophthalmic goitre.

A preparation similar to thyroidectin has been in use on the continent for some time. Dr. S. Christens has used milk (rodagen) and serum of thyroidectomized goats, but for the last four years has confined himself to the use of blood tablets prepared in the following manner: The blood of a thyroidectomized goat is distributed in thin layers in shallow dishes, dried in an incubator at body temperature, powdered, some gum added and made up into 35 cgm. (5 gr.) tablets. These tablets proved readily soluble in artificial gastric juice.

Personal Experience with the Serum Treatment in Exophthalmic Goitre.—Dr. Sydney Kuh reported 11 cases of exophthalmic goitre, of which one was treated by the administration of the serum of the thyroidectomized animal furnished by Merck, while in the other cases either the liquid or the desiccated serum furnished by Parke, Davis & Co. was given.

His conclusions are as follows: His experience has not been sufficient to justify him in making any statement as to the curative effect of the serum. He believes, however, that he is justified in saying that it is an excellent palliative at least; that it is not an infallible remedy is very probably true. Amongst the 11 cases there was one in which the effect of the treatment was temporary only, and another one in which there was possibly no effect at all. One thing was very striking, and that was the marked and rapid improvement in the subjective condition of most of the patients. Within a few days after taking the first dose of the serum they would report a change for the better. The remedy influences the pulse probably as much as anything that is employed in the treatment of tachycardia in exophthalmic goitre; it increases the appetite and decreases nervousness. It has, if one may judge from the series of cases he reported, a very pronounced tendency to increase the weight. Theoretically, it seems hardly probable that the results from the serum should be permanent. One perhaps may expect that after some time a smaller dose may suffice; that perhaps an intermittent treatment only may be required. In one case, the first one reported, the patient has been without serum now for quite a long period, for approximately two and a half years, and has remained in excellent health all that time, but it seems doubtful whether she will remain in that condition unless she occasionally takes a few doses of the serum.

Two Severe Cases of Exophthalmic Goitre.—Dr. Charles L. Mix said he has had two very pronounced cases of exophthalmic goitre since Dr. Moyer put the serum into his possession. The first of these was a woman who was in the Mercy Hospital for quite a period, with no diagnosis. Her case was regarded as a peculiar one. It was recognized that the heart was dilated, but further than that, and further than the neurasthenic and hysterical symptoms, nothing positive was made out. When he returned from his vacation in August or September the interne showed him the woman, and after awhile the speaker recognized the case as one of exophthalmic goitre.

She weighed from 22 to 30 pounds less than she does at the present time. She was toxic in the ordinary sense of the word. She was taken by a visiting nurse to Grove House, and is now working there in a laundry. She has gained forty pounds since she has been in Grove House, and her pulse ranges in the nineties at the present time. In addition to the improvement in tachycardia, in addition to the gain in weight, and in addition to the gain in strength, the doing away of myasthenia, there is a very pronounced effect also in the condition of the thyroid gland. The neck has diminished materially in size. The heart also has decreased in size materially.

The other case was more severe. She had reached the stage when the heart was giving away, when edema was rapidly appearing. She had very marked ascites. The girth of the navel was 37 inches. Her legs were excessively edematous. Her heart was a beautiful example of delirium cordis. The pulse rate ranged between 140 and 150; the neck measured 14½ inches in circumference. She was 20 pounds under weight, but after the disappearance of the ascites she gained 20 pounds. At first she refused to take serum, but was finally induced to take it, and took it in doses of 2 c.c. three times a day, which was afterwards reduced to 1 c.c. three times a day, and then the serum was abandoned altogether and dry serum was given. The other case took the dry serum from the start. She was given thyroidectomized serum, or the serum from a thyroidectomized horse was injected and the patient grew worse, so that she would have nothing to do with horse serum. Goat serum proved to be her salvation. After taking it during the winter the patient was able to come to the speaker's office. She has gained about 20 pounds in weight; her pulse rate at present is about 111.

Dr. Mix said there is one other practical point in regard to the method of administration of the serum. He believes there is a neutralization point in these cases. If he gave too large doses hypodermically, the woman would have marked symptoms of cardiac failure which resembled those of angina pectoris. They were associated with pain in the arm. The danger of collapse was so great on one accasion that the author was telephoned for and certain stimulants were given at the time, and after the dose had been decreased the patient got along better. On the other hand, he thinks that one can give too little. In the other case he has referred to, the one, with ascites, he tried to reduce the dose to ten grains a day. But this was too little, as the patient began to get worse again. She would take fifteen grains daily without any particular difficulty, and he presumes that in the course of time it will be possible to reduce that still further.

We must give enough serum to neutralize the condition, and ultimately we shall have less and less dosage to give, until finally it will be quite easy to run these patients on a very limited amount of serum—maybe give it to them once or twice a week.

Dr. Harold N. Moyer agreed with Dr. Mix that the two cases reported were extreme cases of exophthalmic goitre. Of three cases of his own that were operated upon all died.

As to the word cure, Dr. Mix expressed some doubt. The speaker did not think it was a good remark, as cases of exophthalmic goitre got well without any treatment. If they recover without treatment they should recover from the use of serum sometimes. There is unquestionably a vicious circle in cases of exophthalmic goitre as in other affections, and if one can improve the physical condition of the patient, can control the symptoms, he gives the patient a chance to recover.

Dr. Julius Grinker said he had seen a case that was almost as severe as, if not worse than, the case cited by Dr. Mix, and the patient had gotten well in spite of treatment given by various physicians. From having been reduced to a skeleton and ready for the grave, he had gained in weight and strength so that now he was practically well. Dr. Grinker had seen a few other cases recover after they had discarded all treatment. He believes the benefit derived from the serum treatment is due very largely to rest rather than to the serum.

The ability to lose and gain weight rapidly is almost pathognomonic of Graves' disease, so that a temporary gain in weight is no criterion of successful treatment.

Dr. Grinker quoted from Eulenburg, who says that he has gained the impression that anti-thyroidin does not possess specific effects, either curative or ameliorative. Eulenbug speaks very discouragingly of the operative treatment in this disease.

Dr. Moyer had stated he thought the serum could be a diagnostic aid, and that whenever a patient does not improve on serum the diagnosis of exophthalmic goitre was probably wrong, but he had said that some of his own patients had gotten well without any treatment. He thought Dr. Mix was too enthusiastic, and had seen severe cases get well without treatment. He referred to the work of R. Breuer, who showed that iodine given internally in the form of the iodides or applied externally may produce exophthalmic goitre, which will persist long after the discontinuance of the drug.

Dr. L. L. McArthur said it would be a mistake to convey the impression that every case of thyroid enlargement, associated with peculiar nervous phenomena, is necessarily fatal if operated upon. He made a plea that cases of exophthalmic goitre be turned over to the surgeon before they are desperately ill or in the last stages of the disease.

Dr. C. C. O'Byrne said that sometimes there is a single nodule in the thyroid gland very firm, and this causes the symptoms of Graves' disease, perhaps as commonly or more so than a diffuse enlargement. He cited two such cases, one a medical student, who consulted him with reference to a small lump on the inside of the neck. The symptoms complained of were those of extreme nervousness, wakefulness, increase in the size of the gland, dyspnea on exertion, a well-marked tremor, and pulse 100, but no exophthalmos and no apparent struma. The single nodule was about the size of a walnut, and was situated in the lower part of the thyroid gland on the left side. This nodule was removed, and followed by relief of all symptoms.

The other patient, also a student, had a uniform enlargement of the thyroid gland, soft and pulsating, and a pulse exceeding 100. He was very nervous, had tremor of the hands and lips and disturbance of vision. He was put on the serum treatment, but no improvement in symptoms was observed from its use. Dr. O'Byrne operated, removed the nodule and the symptoms disappeared.

Dr. Moyer, in closing the discussion, said he did not wish to convey the impression that surgery was valueless in cases of exophthalmic goitre. He simply mentioned the fact that three of his operative cases died. These were desperate cases. Surgery is undoubtedly of value in suitable and selected cases.

THE BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

March 16, 1905.

The President, Dr. Morton Prince, in the Chair.

Spinal Tumor.—Dr. Walton reported two cases of operation for spinal tumor. The first case (seen in consultation with Dr. Painter) was a man of 49, who had had pain for two years in the arms, legs and shoulders, severe for two months before the operation, requiring one-half grain of morphia a day; he was losing weight rapidly. The pain was localized at about the level of the sixth cervical vertebra and extended into the left arm, involving the shoulder, movements of which increased the pain. There was paresthesia and anesthesia in the arm, especially involving the ulnar side. Operation by Dr. Munro revealed myeloma of the laminæ. The laminæ (seventh cervical and first dorsal) was found to extend not only into the muscles, but so as to press upon the dura. The growth was curetted out. No narcotic has been required since the operation. The pain in the arm practically disappeared within two weeks, and the sensation and strength were improving. There has been no recurrence of pain during the subsequent three months.

The second case was seen in consultation with Dr. Paul. A man of 53 had complained for several months of pain between the shoulders and down the arms, with some dull pain in the lower extremities, with paresthesia. The power in the legs gradually lessened, and after his admission to the Massachusetts General Hospital he became bedridden. There was a modified Brown-Séquard paralysis. The loss of motion predominated on the right, sensation on the left. There was weakness in both hands and spasticity with Babinski first in the right, then in the left leg, and anesthesia to the level of the xiphoid.

Operation by Dr. Harrington revealed an aparently healthy cord. The laminæ of the seventh cervical and first dorsal were removed. The patient died the day following operation, and a firm fibrous tumor was found at the autopsy (by Dr. Oscar Richardson), an outgrowth from the intervertebral disk between the seventh and first dorsal vertebræ. This had pressed upon the anterior surface of the cord, producing a deep indentation without causing destruction of its tissue.

Dr. E. W. Taylor, in connection with the very successful diagnoses made in Dr. Walton's cases, mentioned two instances in which no tumor was found at operation in spite of the presence of supposedly characteristic symptoms. Both of these cases were carefully studied and presented signs and symptoms pointing to lesion of the spinal cord. In the first there was bilateral intercostal pain, with succeeding disturbances in the lower extremities, both of motion and sensation, together with certain sphincteric disturbances. The height of the lesion was apparently easily localizable, but the operation, which extended over several vertebræ, showed no external abnormality either in or about the cord. The patient's condition remained unchanged, except for an increased spasticity and the relief of the intercostal pain due to the cutting of the dorsal nerve roots. The second case presented the classical symptoms of a tumor in the cervical enlargement, with extreme pain running down the arms and the ordinary symptoms in the body and legs. Here also operation revealed nothing abnormal. The patient died shortly after, but no autopsy was obtained. These two cases were reported as illustrating the uncertainty of diagnosis, even in cases where the symptoms seemed unequivocal.

Dr. Brownrigg said that the difficulty sometimes of locating definitely an apparent transverse lesion of the cord led him to present the following facts in regard to a case of spastic paraplegia with some involvement of the arms and total paralysis of the sphincters.

There was probably a history of syphilis some years before the onset of the paralysis, which progressed slowly and continuously for several years before before the end stage of spastic rigidity in which he was first seen. The symptoms and their progress were very typical of that form of paraplegia separated by Erb as distinctive of syphilitic lesion in the dorsal cord. Indeed, there had been so many localizing symptoms than an operation was suggested by some to see if relief could be given from the possible tumor.

This was not thought advisable, on account of the probable diffuse character of the lesions and the end stage of the disease, rather than from any doubt as to where the causal lesions were situated. There were no marked mental symptoms, though towards the terminal weeks of his life there was spastic irritability of his facial muscles and of others controlled by the cranial nerves.

The autopsy revealed, besides atrophic spinal degeneration, multiple cysts of clear fluid under the pia over the cortex corresponding to the leg center, and forward over the anterior lobes with other local thickenings of the pia over the motor areas. These were so marked that it was somewhat surprising there was not more mental involvement or other symptoms usually accompanying cerebral cysts.

The pathological examination had not been completed, but the spinal degeneration apparently was not dependent upon local pressure on the cord, but upon a descending degeneration from pressure on the cortical centers.

A Case of Juvenile General Paralysis.—This was presented by Dr. P. C. Knapp, The patient, a boy 13 years of age, had a very clear history of hereditary syphilis. The father said he had been infected through sucking another man's pipe, took much medicine, and put off marriage for six months. The first child lived only a day or two, the second was still-born and had been dead several weeks, and this child was the third. He was well for three months, and then was very sick until the age of two with snuffles and a skin trouble, of which he now bears traces, many circular, white, punched scars over the left loin and flank, extending to the right loin. He has also linear cicatrices at the corners of his mouth, but no keratitis or Hutchinson teeth.

He always wanted his own way and had a bad temper. He went to school, and up to six months ago was brighter than the average. Two years ago he had an emotional shock, and about the same time an attack of zoster. His father had noticed some change in disposition since then, but it had been marked only in the last six months, although last June his school work was so poor that he was put back. There had been a notable failure of memory, especially for his school work and prayers. He forgot what he was sent to do when he went on an errand, but he remembered past events fairly well. He was irritable, quarrelsome and violent. He had never had headache or convulsions. He had had some trouble in swallowing solid food toward the end of a meal. For five months there had been more or less incontinence of feces. His speech was indistinct and he ran the syllables together. He used to write quite well, but now his handwriting is very indistinct. He counted fairly and repeated the days of the week, but gave the months "January, February, July, Monday," and the alphabet "A, B, C, D, F—I can't say it." He was very obstinate and often insisted that he could not say what he had just said. He failed to do simple examples, and then repeated more difficult parts of the addition table correctly. There was no tremor of the face, lips, tongue or hands. The ankle-jerks and knee-jerks were absent. The right pupil was a trifle larger than the left; both reacted sluggishly to light, the right reacting very little. The reaction to accommodation was fair. Four weeks ago Dr. Greenwood found the discs congested and the outlines blurred, a "slight papillitis," which had improved considerably.

Part of the failure to respond to mental tests seemed to be due to obstinacy rather than to dementia, but there was also considerable mental failure in the case. Slight degrees of neuritis have been spoken of as occurring in general paralysis, chiefly by earlier writers. Mendel, Schüle and others, but recent writers, Bullet and Blocq, Wollenberg, Ziehen, Bianchi, Tanzi, Weygandt, Dupre and Kraepelin have little to say about it. Dr. Knapp had never seen this condition before although atrophy is not very unusual. The optic nerve, the incontinence of feces and the trouble in swallowing had improved under iodide.

The reported cases of juvenile general paralysis are constantly increasing in number, so that it is now not so very rare. The condition of the pupils and the tendon reflexes, and the handwriting, added to the mental symptoms, make the diagnosis tolerably assured.

Dr. Prince reported a case of tic convulsif and showed the patient.

Dr. Walton called attention to the obsessive tendencies of Dr. Prince's patient in other directions than in the movements. Such tendencies were shown, for example, by pronounced inclination to return for reassurance after having locked doors, turned off the gas, etc. Later Dr. Walton suggested that tic obsessif would be a more appropriate term for these cases than the more misleading tic convulsif at present in use.

Dr. Baldwin read on "Some Cases of Hysteria." One patient was shown who had recovered from hysterical aphonia and paraplegia of long standing.

Periscope

Archives de Neurologie

(Vol. 19, 1905, No. 110, February.)

1. Sexual Excitation in the Anxious Psychopathies. CULLERRE.
2. A New Case of Conjugal Paresis of Syphilitic Origin. CARNIER and SARTENOISE.
3. Epilepsy, Alcoholic Delirium, Melancholia with Suicidal Attempts and General Paralysis in the Son of an Alcoholic Mother and a Father who Suicided, the Patient Himself being a Syphilitic and an Alcoholic. SIMON.

1. *Sexual Excitation in the Anxious Psychopathies.*—A lengthy article dealing with the relationship between the anxiety phobias and sexual excitation. Space forbids an extended consideration. There is at first an association of the two symptoms. Sexual excitation is a common phenomenon in all mental maladies, observable often in the periods of invasion not only organic, but of simple psychoses. It is frequent in the course of the systematized delusions and responsible for many of the delirious ideas, but it plays only an accidental rôle. Predisposition and heredity and emotional desequilibration have their influence. The author thinks there is a pathogenic bond between the two conditions. The two symptoms are generated by an incidental mechanism. Freud's theory which makes the anxiety a condition consequent upon an accumulated genital tension is not favored by the author. In his cases the anxiety was always the primary phenomenon, then the sexual excitation. Experiment has shown the existence of cortical centers governing the functions and appetites, and in particular the organs of generation, and the author traces the psychologic connection between erotic ideas and sexual excitation, the persistence in consciousness of erotic ideas, but he does not trace the relationship between these and anxiety. There is also a discussion of relative control of the superior cortical centers and the automatic infracortical centers, but no explanation is essayed by the author, he desiring merely to show the coincidence of two phenomena which would seem to exclude each other.

2. *A New Case of Conjugal Paresis of Syphilitic Origin.*—Cullerre had previously reported three cases in which, however, the luetic etiology was not unqualifiedly established. In 1904, Cullerre reported a fourth case, in which he thought himself entitled to say: "I consider the last case as favoring the luetic origin of general paralysis." The two writers report a new case further fortifying this position. This case, with those of Cullerre (1890) and cases of conjugal paresis on a luetic basis previously reported by Acker in 1887 and Mendel in 1888, establish in the author's opinion the contention of this article. There had been about forty cases reported up to 1904.

3. *Epilepsy, Alcoholic Delirium, Melancholia with Suicidal Attempts.*—The history of this young man is carefully traced. It shows the unusual association of symptoms of very distinct entities. The influence of the alcoholic and the luetic heredity is discussed. This case must be read in the original. WOLFSTEIN (Cincinnati).

(Vol. 19, 1905, No. 111, March.)

1. Secondary Degeneration after Focal Softening in the Calcarine Region. PROF. WEBER.
2. Clinical Study on Stereotypy in Dementia Precox. DROMARD.

1. *Secondary Degeneration after Focal Softening in the Calcarine Region.*—Patient died aged 76 years. Admitted profoundly demented, complete amnesia; no history obtainable. Visual acuity much diminished, the glance is vague. Patient did not fix the persons addressing her. She winked, however, when an object was approached to the eyes, and infrequently she could recognize what was shown her. The dementia precluded any careful analysis. Autopsy.—A focus of softening was found in the right calcarine region, about 40 mm. long. Pal stain was used. The examination was rendered more difficult by reason of an unfortunate cut made during autopsy precisely through the region of the external geniculate bodies. The author agrees with the well-known view of v. Monakow that every destruction of the median part of the occipital lobe must cause a secondary degeneration of the cortical visual neurons. C. Genic Ext. optic rad. cortex. There were found in Weber's case besides the softening in the calcarine region (1) slight areas in corpus callosum, (2) a linear destruction in the hippocampal convolution, (3) one in the convolution gendronnée. All of these were "quantités negligeables." The drawings accompanying the article give a clearer idea of the secondary degeneration. The article deserves careful perusal as a contribution to the study of hemianopsia after focal lesions in the visual area.

The microscopical examination was made with great care, but it is a pity that control specimens with the Marchi method were not made. We give Weber's résumé: Following softening in the calcarine region there was observed: 1. The sagittal fibers were directly injured equally. 2. The secondary degeneration was at first localized in the inferior part of the Optic Radiation and the Fascia Long Inf. 3. This degeneration could be subdivided into three areas: (a) More frontal—an inferior zone tending to the temporal lobe; (b) a superior zone going to C. Genic Ext.; (c) one to Pulvinar. 4. This degeneration was certainly more extensive in F. L. I. than O. R. Weber also concludes, in agreement with v. Monakow, that the dorsal portion of the sagittal fibers of the occipital lobe are connected with the posterior areas of the thalamus opticus and with the parietal cortex. They have no relations of any importance with the cuneus lobuslingualis or O2. After v. Monakow, therefore, the bundle which unites the visual cortical area with the primary optic centers divides at the height of the C. Genic into three portions: (1) To the area of Corp. Quad. Ant., (2) to the pulvinar, (3) to the C. Genic Exter.; 2 and 3 were degenerated. Weber demonstrated also that the lateral inferior white matter of C. Genic Exter. was intact, and probably not in relation with the optic sphere. As regards the function of F. L. I., Dejerine's view is "that this inferior longitudinal bundle is an association bundle connecting the occipital lobe, and especially the visual sphere, with the temporal lobe." Monakow and Sachs agree with Dejerine. Flechsig (with his usual methods) and Probst (based on study of lesions in man and animal experimentation) view the F. L. I. as a projection bundle connecting the Thal. Optic with the visual cortical zone. Sterokotlitzki believes that the F. L. I. is partly an association, partly a projection system: (a) The inferior portion connects the occipital to the temporal cortex (associative); (b) the superior goes to the infra-cortical centers. Thal. Op. Pulv. C. Genic Ext., C. Genic Int. and Nucleus Lentic. (projection). With slight modifications, Weber agrees with the latter view. Weber is of the opinion that the localization of the secondary degeneration depended without doubt upon the lesions of the fibers extending from the cortical focus.

2. *Clinical Study on Stereotypy in Dementia Precox.*—This is a very long classification of various types of stereotypy, both of attitudes as well as of various other forms of motorial activity, such as speech, writing, mimicry, the gait and complex acts. There is also a consideration of the evolution of stereotypy. Kraepelin defines this form of activity as the abnormal duration of motor impulses which produce a permanent contracture of certain muscle groups, or a repetition of such a movement. The term contracture as used here in the spirit of the author would seem to indicate that physiological state presided over the symptom. The direct modification of cerebro cellular activity under the influence of some presumed toxin conditioned the morbid process, as shown by the prolonged attitudes or repeated movements of the subject. Others, particularly Cahen, would rather consider these states as not being convulsive in character, but as actions which were at first voluntary and conscious, and which later become automatic and subconscious by this fact of long duration and repetition. Here we are not concerned with an active manifestation, with the phenomenon of irritability belonging to the domain of primitive automatism, but it is a residual manifestation of a mind habit belonging to the domain of secondary automatism. We are, therefore, in the presence of a problem in every way comparable to the distinction between the tics and spasms, a distinction well cleared up by Meige and Feindel.

It would also appar to be necessary to distinguish with as much precision as the nature of so complex a subject permits between active manifestations and those of the subconscious residual period. The writer bases his views on the microscopical researches of Klippel, who describes first the immediate lesions in which there is a granular degeneration of the cells, and so-called consecutive lesions, which are characterized by the destruction of connections between different territories or the different cells of the cortex through atrophy of their prolongations, which bring about intercommunication. The article is not well adapted for review. The author also discusses the value of stereotypy as a diagnostic sign, and is not inclined to look upon it as specially peculiar to dementia precox, being found in other forms as well. The prognostic significance, as well as the relation between convalescence and the disappearance of stereotypy, are also discussed. Wolfstein (Cincinnati).

Nouvelle Iconographie de la Salpetriere

(Mars-Avril, 1905, No. 2.)

1. Friedreich's Disease and Hereditary Cerebellar Ataxia. Raymond.
2. Alterations in the Cerebral Tissue Due to the Presence of Tumors. Weber, Papadaki.
3. Syndrome of the Peduncular Calotte. Gruner, Bertolotti.
4. A Case of Acromegaly with Hyperplastic Lesions of the Pituitary Body, Thyroid Body and of the Suprarenal Bodies. Ballet, Laignel-Lavastine.
5. Cyanosis of the Retina in Sclerosis of the Pulmonary Artery. Babinski, Mlle. Tufesco.
6. Acute and Circumscribed Chronic Edema of Nervous Origin. Valobra.
7. A Case of Early Thoracic Deformity Following an Acute Pleurisy. Maitignon.

8 Professional Cramp and its Treatment by Massage and Reëducation. Kouindjy.

1. *Friedreich's Disease.*—Raymond's conclusion from the study of these cases and from the pathological findings in one of them is that it is practically impossible to ascribe all cases of this symptom complex to one morbid entity; that is, to an isolated atrophy of the cerebellum. The

different clinical pictures represent lesions in the cerebellum of different kinds and of different localizations; sometimes in the centers themselves, and sometimes in the efferent or afferent paths. There is little advantage in multiplying the different types of this complex, but it is advantageous to find out just what systems are involved and to translate them in the anatomical terms of their localization.

2. *Tumors and Cerebral Tissue.*—This work was undertaken at the suggestion of v. Monakow. The plan of the work is to describe the effect on the brain tissues due to the tumor itself in five cases of brain tumor of different sizes and localization. The observations are divided into two categories: 1. The tumor develops at the expense of the cerebral tissue. 2. The neoplasm has its point of origin outside of the brain, which it compresses in its development. Some of the results of this study are as follows: There appears to be a lymphatic current from the surface of the brain to the ventricles. The part of the brain peripheral to the tumor suffers the most, and not by the alteration of the circulation of the blood, but by that of the lymph. This current appears to take its course across the aqueduct of Sylvius toward the subdural space of the spinal cord. The consequences depend upon whether the tumor intercepts the current or not. The intracerebral pressure is not equal throughout, if that can be judged by the alteration in the tissue. It has its maximum of intensity at the periphery of the tumor if the ventricles are dilated in its neighbborhood. Under the influence of an augmentation of intracranial pressure the convolutions at first become closely pressed together, forming fissures, then the tissues become more dense and their outline is effaced. This is the first stage. Later the cerebro-spinal fluid incompressible causes atrophy of the tissues. The tissue in the white matter shows more alteration than that of the cortex. In the white matter the association fibers suffer more than the projection fibers. When a unilateral tumor obstructs the ventricular circulation it is the rule that the opposite ventricle should be more dilated. Equilibrium is thus established. Dilatation of the perivascular spaces, formation of hemorrhages, appear to accompany regularly the development of tumors. Although the lymphatic circulation is necessarily closely allied to that of the blood, it cannot be admitted that the lymphatic stasis should be simply the consequence of the venous compression. There is probably a special mechanism of nutrition of the brain of which we are at present in ignorance. The author does not believe that the toxic theory advanced by Brissaud and Souques explains even in part the pathological phenomena of cerebral tumor.

3. *Peduncular Calotte.*—Raymond and Cestan reported in 1903 three cases in which there was a symptom-complex due to lesion in the superior protuberential region. It is characterized on one side by a paralysis of the lateral movements of the eyeball, and on the other by a hemiplegia of the arm and leg, affecting very slightly the motor strength, but showing itself on the contrary by tremor, incoördination, athetoid movements, by cerebellar asynergia and by subjective and objective disturbances in sensibility. This peculiar group of symptoms, fixed in type, was found in all three cases to be due to a solitary tubercle at the level of the protuberential "calotte" behind the level of the sensory fibers under Wernicke's crossing, under the knee of the facial in the region extending between the nuclei of the third and sixth pair. Two cases are described by the author, one of which came to autopsy. In the first a diagnosis of probable tumor in the neighborhood anterior quadrigeminal bodies was made. In the second case at autopsy, a tumor, a tuberculoma in the central space of the "calotte" involving the grey substance of the aqueduct obstructing the central canal and destroying the nucleus of the third pair on both was found. An area of softening extended to the internal and superior half of the sensory fibers, destroying the anterior left quadri-

geminal body. The findings in these cases are carefully discussed in the light of the anatomical and physiological logic of the symptoms.

4. *Acromegaly.*—A case of acromegaly in a woman 70 years of age. Clinical résumé: Typhoid fever, menopause, cyphosis and progressive deafness, hypertrophic deformity of the head, hands and feet, ictus, right hemiparesis, flaccid paraplegia, chronic aortitis with insufficiency valvular. Pulmonary terminal congestion. Death. Autopsy showed parenchymatous hypertrophy of the hyphophysis, cirrhotic hypertrophy of the thyroid gland, cirrhotic superenal hypertrophy with adenoma, calcareous granulations of the choroid plexus.

5. *Cyanosis of Retina.*—A report of two cases and a résumé of the literature..

6. *Chronic Edema.*—Continued paper.

Thoracic Deformity.—Not adapted for abstract.

8. *Professional Cramp.*—An acocunt of the use of certain exercises in the treatment of these conditions. S. Schwab (St. Louis)).

Journal de Neurologie

(1905, No. 4.)

1. The Spinal Localization of the Muscles of the Perineum and the Rectum. S. Irimesco and C. Parhon.
2. The Physiopsychology of the Nuns of Port Royal. C. Binet-Sangle.

1. *The Spinal Localization of the Muscles of the Perineum and Rectum.*—The authors, in an examination of two cases dead after extensive suppuration involving the perineum, anal region and rectum, found a group of cells situated in the second and third sacral segments in the mid-portion of the anterior horn, behind and slightly to the inner side of the group X of Onuf (the cells of which group these cells somewhat exceed in size) in reaction. The intermedio-lateral group in the third and fourth sacral segments also showed some changes. From a consideration of these cases and comparison of them with the findings reported by other investigators, they conclude that the perineal muscles are represented in the cell group first mentioned (adjacent to group X of Onuf), and that the intermedio-lateral group belongs to the sympathetic and has to do with the innervation of the unstriped muscular fibres of the rectum, while a small group just inside the intermedio-lateral group, also found in the reaction, they regard as the probable center for the internal sphincter of the anus.

2. *Physiopsychology of the Nuns of Port Royal.—A seventh* series of five cases from the famous Jansenist community. From an analysis of ten the author finds the following: One belonged to a family showing signs of advanced degeneration. In two there were various signs of degeneration, in one hypermotivity and tendency to melancholy, in all hyper-suggestibility. In two the cause of death could be ascertained, one dying from pneumonia, the other from cancer. The average age at death in four cases was 67 years.

(1905, No. 5.)

1. On the Presence of a Special Network in the Pigmented Region of the Nerve Cells. G. Marinesco.
2. The Pathogeny of Conjugate Deviation of the Eyes and Head. Deray.

1. *Special Methods in Pigmented Region of Nerve Cells.*—The author has studied the fibrillary network in the nerve cells under a number of different conditions, by the fibril method of silver impregnation of Ramon y Cajal. In middle-aged and elderly persons or animals, where the cells are apt to contain pigmentary deposits, he finds in the pigmented region a special form of fibrillary network with meshes of very thick fibrils which show up darker in color than the fibrils of the

other part of the cell, forming what he calls "the black network." The appearances vary somewhat in cells from different regions. In a type of cell in which the fibrils alone are prominent he finds no network in the pigmented region, but the fibrils become much thicker and darker there, and when they have passed this zone they resume their normal proportions. In the cells of the spinal ganglia in certain diseases, as lepra and pellagra, the author has found a network which he thinks identical with that occurring in the pigmented region as described above. This network is very resistant to different agents hurtful to the normal cell. As to its origin, the author thinks that it is derived from the ordinary net work through some chemical change closely connected with that producing the pigmentary deposit. Discussing the effect on nervous action of this change, while admitting that we have no definite information upon which to construct a theory, he suggests that possibly the wave of specific nervous energy is in some way diminished in intensity by passing through the areas of thickened fibrils. He has found "hypertrophy of the neurofibrils" so frequently in cases of rabies that he is inclined to consider it as a lesion characteristic of this disease, and quotes a recent work of Ramon y Cajal and Garcia as strongly supporting this view.

2. *Conjugate Deviation of Eyes and Head.*—Bard, and after him Dufour, have sustained the opinion that conjugate deviation is sensorial in origin, and often due to concomitant hemianopsia. This view the author combats, bringing forward the evidence opposing it. He calls attention to the fact that lateral movements of the head and eyes are connected not only with visual but also with auditory impressions, and that conjugate deviation is known to be produced by excitations arising in a number of different regions of the cortex. From an array of cases he shows that conjugate deviation and hemianopsia are by no means always associated. Considering the subject as a whole, the author concludes that conjugate deviation is not necessarily connected with hemianopsia, but may be due to lesions in the cortical centers for eye and head movements, in the parietal or frontal association centers of Flechsig, or in the association fibers connecting these with the visual centers; possibly also in the basal ganglia. In his opinion the location of the lesion in the posterior center of the parietal region explains the symptoms best. If it is destroyed the patient looks toward, if it is irritated, away from, the lesion. ALLEN (Trenton).

Psychiatrisch-Neurologische Wochenschrift

(May 6, 1905.)

Contains only articles of local interest.

(May 13.)

The Ceni Method of Treating Epilepsy.—Ceni's method of treating epilepsy by serum injection advocated by him in 1901 did not attract much attention in Germany. The author, however, is of the opinion that the result he claimed should not go unnoticed. One case he reported was of a young girl of 12 years of age, who had suffered from epilepsy for three years, having about fifty attacks a month. She was greatly emaciated, and apparently demented. As a result of the treatment she was soon free from attacks, improved mentally, and took on weight in a short time. In fact, "she showed a complete revolution in both physiognomy and character." Ceni's procedure, described by Wende in a previous number of the *Wochenschrift*, consists in the injection of serum taken from other epileptics or from those being treated,gradually increasing the dose from 72 to 410 g. in a period of from two to six months. Wende's results were not good, and he says himself that he was hindered from obtaining as good results as others by conditions he could not control. He stopped the bromide his patients were taking and begun the serum treatment after two weeks. He treated twelve men, most of whom showed some improvement, and two also a change in the psychical state. The results of stopping the bromide were difficult to determine and to differentiate from those of the serum. In this connection Holme's interesting figures are quoted: Twenty epileptics had with bromide in eight months 1,913 attacks; in eight months after the withdrawal of the bromide 1,976 attacks, and in the following eight months 1,499 attacks.

The author reports three cases. They all had the bromide withdrawn for from seven months to two years and their manner of life and food was not changed. Treatment in these cases was practically nil; the convulsions were not decreased nor the mental state improved.

As a result of these cases and also of Wende's failures, the author concludes that Ceni's theory that there is an autocytotoxin and an anti-autocytotoxin in epileptic blood serum is without clinical evidence to support it. WHITE.

Miscellany

TUBERCULOUS MENINGITIS. W. F. Cheney (Journal A. M. A., May 20).

Cheney reports and comments on three cases of tuberculous meningitis with special reference to the diagnosis. He mentions the indefiniteness of the early symptoms, and says that we should always be suspicious of tuberculous meningitis in a child in illness with associated digestve disturbances, slight fever and irritability of temper. Stupor is more significant, but it may also be due to intestinal toxemia or some acute infection, but in such case there is usually a higher fever. Evidences of intracranial pressure, such as pupillary abnormalities, local paralyses, irregularities, etc., are still more definite indications, but these, like the mental condition, may vary from day to day, thus giving rise to false hopes of recovery. Rigidity of the neck muscles is a very valuable sign, only likely to be simulated by disease of the cervical vertebræ or rheumatic torticollis, and in these other symptoms of meningitis are apt to be wanting. The Kernig sign, so valuable in the adult, is, according to Cheney's experience, difficult to elicit in children, owing to their fear of manipulation. The white blood count is of value, a moderate leucocytosis pointing to tubercular rather than to other forms. Lumbar puncture furnishes negative evidence of value, with the other symptoms present, a clear fluid, free from micro-organisms is characteristic of tuberculous meningitis. One of his reported cases is of interest on account of its occurrence in an adult, its clinical resemblance to cerebrospinal meningitis, and from its being the only one of the three in which an autopsy was obtained. We are apt to associate tubercular meningitis with infancy, but it can be excluded at no period of life. In this case the low leucocytosis and the lumbar puncture findings confirmed the diagnosis of tuberculous meningitis during life, and the autopsy revealed a preëxisting lung focus. Tuberculous meningitis is always secondary to the disease somewhere else in the organism. The article concludes with remarks on the differential diagnosis from other conditions, and especially from the other forms of meningitis.

A CASE OF MIND BLINDNESS UNIQUE IN THAT THE ENTIRE MESIAL SURFACE OF BOTH OCCIPITAL LOBES AND BOTH OPTIC RADIATIONS WERE PRESERVED. Ward A. Holden (The American Journal of the Medical Sciences. May, 1905).

To sum up in the words of the author, this was a case of dementia after hemiplegia, together with aphasia, apraxia, and an interference with vision which much of the time amounted evidently to total blindness. The retinæ and optic nerves were normal. In the brain there were found nearly symmetrical areas of softening in each hemisphere, including the angular and supramarginal gyri, and reaching back on each side nearly to the tip of the occipital lobe, caused by the occlusion of the parieto-temporal branch of each middle cerebral artery from the point where it was given off. The calcarine cortex, optic radiations, and optic tracts were normal. The case shows that extreme disturbance of vision may result from interference with the higher cortical visual centers alone.

C. D. Camp (Philadelphia).

A Case Simulating Intracranial Tumor in which Recovery was Associated with Persistent Cerebrospinal Rhinorrhea. T. R. Glynn and E. E. Glynn (The British Medical Journal, April 22, 1905).

The first symptoms developed after a traumatism to the top of the head, which caused a temporary loss of consciousness. For the next three years he had recurrent attacks of headache, about every two weeks, associated with vomiting, giddiness and depression, and double vision on looking toward the left. He suffered constantly with failure of memory, lack of energy and some giddiness, any attempt to stoop causing him to fall forward. On examination he was found to have weakness in external rectus muscle, an optic neuritis, exaggerated knee-jerks and a marked left hemianalgesia. In the next year he failed gradually so that there was a distinct failure of muscular power of the extremities and tremor of the hands. He became demented, had three epileptiform convulsions in the year, and developed incontinence of urine and was nearly blind. When in this condition clear fluid commenced to escape in drops from the right nostril and he immediately began to improve. His mental vigor and muscular strength returned and his sight was restored. A year later he was almost completely restored to health. The optic neuritis and all other signs of cerebral mischief had disappeared. A careful examination of the fluid showed it to have all the physical and chemical characteristics of the cerebrospinal fluid. It was excreted at the rate of about seven ounces in twenty-four hours, a less amount than in any other reported case.

C. D. Camp (Philadelphia).

A Study of Acute Hemorrhagic Encephalitis. E. E. Southard and C. W. Keene (The American Journal of the Medical Sciences, March, 1905).

Six fatal cases in man were examined, all but one cases of general infection with the *staphylococcus aureus*. A history of antecedent disease was the rule. The syndromes, which were chiefly of sudden onset and rapid course, were pyemic, meningitic or cerebral in type. The slower course gave more cerebral symptoms. The type of inflammation produced by the *staphylococcus aureus* in man is one in which hemorrhage is a prominent feature. The site of election for the hemorrhagic lesions is the subcortical region supplied by the long or medullary branches of the cortical vascular system, and they vary in appearance from red softening or multiple ecchymosis to frank and sometimes voluminous hemorrhage. Experiments on guinea pigs carried out at the same time showed that the *staphylococcus pyogenes aureus* produces in their brains an inflammatory process, which is shown as a meningitis, ependymitis, or exudation into the brain substance which tends to subside in a limited period without producing hemorrhages, except miliary perivascular ones. Perhaps, also, there are in man cases of encephalitis which reverse themselves and may be mistaken for "functional" diseases and furnish "functional" symptoms during and after tissue repair. C. D. Camp (Philadelphia).

An Investigation on the Regeneration of Nerves. Basil Kilvington (The British Medical Journal, April 29, 1905).

After reviewing the literature on regeneration of nerves, the author details his own experiments which were made on the popliteal nerves of dogs. The results of the reunion after division, and suturing in various combinations, such as the central end of the internal popliteal to the peripheral portions of both internal and external popliteal nerves, are investigated both from an anatomical and physiological standpoint. The author summarizes his conclusions as follows: "It is possible to function the two opposing groups of muscles by a single nerve, which previously supplied one group only. Second, when the central end of one nerve is joined to the peripheral ends of two nerves there are many more fibers in the peripheral nerves than in the central nerve, so that the nerve fibers in the proximal trunk divide on going to the distal trunks. Third, in some cases, at least, some of the branches from one nerve fiber go to supply one set and others the opposing set of muscles. This may prevent very delicate movements being restored. Fourth, after this form of suturing the arrangement of the nerve fasciculi in the peripheral nerves is considerably altered. C. D. CAMP (Philadelphia).

CEREBRAL SYMPTOMS IN MEASLES. Guy J. Branson (British Medical Journal, April 29, 1905).

The case appeared to be a simple, uncomplicated attack of measles, but on the next day after the appearance of the rash there were noted frequent twitchings of the face and limbs, and the day following a severe general convulsion accompanied by unconsciousness. There were no paralyses or localizing symptoms, but the attack was followed by intense restlessness and cerebral irritability. All the symptoms completely disappeared as the measles rash and fever cleared up.

C. D. CAMP (Philadelphia).

DISSEMINATED SYPHILITIC ENCEPHALITIS. Albert M. Barrett (The American Journal of the Medical Sciences, March, 1905).

A case of brain syphilis occurring in the secondary stage of the disease. The symptoms were those of gradually progressive dementia accompanied by ocular symptoms and general spasticity of the limbs, which was more marked on the left side. Death occurred in coma, and at autopsy the greater affection of the left side was explained by the discovery of a small hemorrhage in the right side of the pons. A detailed histological examination of the brain revealed areas of acute localized encephalitis not apparently connected with the meningitis, which was also present. There was also found a typical diffuse encephalitis, syphilitic arteritis, etc.

C. D. CAMP (Philadelphia).

REGENERATION OF NERVES. Mott, Halliburton and Edwards (Proceedings of the Physiological Society, March 19, 1904).

These authors have followed their studies in nerve degeneration with others in nerve regeneration. Early in the degeneration of the nerve fibers the neurilemma cells multiply, and later seem to assist the phagocytes in the removal of the broken-up myelin drops. The neurilemma cells are found in elongated strands concealing the newly formed axis cylinder. The authors conclude that this cylinder proceeds alone from the central end of the divided nerve, and that the peripheral cells simply act as a scaffolding for it. They call attention to the fact that histological evidence is not sufficient to establish the identity of a nerve fiber; that a strand which looks like one is not really such unless capable of responding to excitation and conducting nerve impulses. The peripheral theory of nerve regeneration has been held to be supported by clinical evidence, on the ground that after freshening up and suturing a divided nerve, sensation returns within forty-eight hours. A case is noted, however, in which the patient stated shortly after the operation that he was able to feel, but these sensations disappeared again in a short time and feeling did not really return for several months. It is suggested that the stimulation of the "freshening" lasted for several hours, the sensation being referred by the patient to the original terminals of the fibers. In certain of the experiments nerves were cut and the upper end covered with a cap so as to prevent the peripheral end from uniting with the central. After 100 or 150 days electrical stimulus found no response under anesthesia even to strong Faradic currents, and the microscope discovered no trace of regeneration. Another significant experiment consisted in dividing and then suturing together a large nerve in monkeys and cats. Upon exposing this nerve after function had been restored it was found united and excitable both above and below the union. A piece of nerve removed an inch below the point of original division was found to consist of fine new fibers with no trace of degenerated products. After ten days the animal was killed and the peripheral portion of the nerve both above and below the second incision was found quite unexcitable and exhibiting Wallerian degeneration. The authors have concluded from some evidence which they offer that the new fibers are less perfectly developed farther from the original point of section. JELLIFFE.

POISONING BY METHYLIC ALCOHOL. C. Ströhmberg, (St. Petersburg med. Woch., Nos. 39 and 40).

During the temporary suspension of the sale of vodka in Dorpat while mobilization was in progress 18 cases of poisoning, 15 of them fatal, occurred from the drinking of a popular vulnerary, also used internally, known as "Kuntzen's Balsam." Although none of the preparation used in these cases could be gotten for analysis, there is no doubt that the poisoning resulted from the substitution of methylated spirit for rectified in the formula of this "balsam." Two cases are reported typical of acute amblyopia due to retrobulbar neuritis. Of the drugs which may produce retrobulbar neuritis methylic alcohol is the most powerful, and in the cases under consideration the other drugs were excluded as possible factors by the small quantity of the preparation ingested, and by certain definite differences in the course of the intoxication. As the percentage of methylic alcohol in the balsam was not known, the fatal dose could not be determined, but if, as seems probable, the percentage was the same as that of ethylic alcohol called for by the formula, fatal results must follow a dose exceeding 35 gm., and serious results may be produced by as little as 7½ gm. The author formulates the symptomology of methylic alcohol poisoning as follows: Immediately after ingestion a bitter, burning taste which may extend to the pharynx and esophagus and be felt behind the sternum; great depression and anxiety; apathy; heavy, aching limbs, headache and vertigo. At first the patient can pull himself together and overcome the weariness, somnolence and tendency to shiver. This may be in slight cases the turning point toward recovery, but in serious cases the shivering may give place to pronounced rigors, and the somnolence to deep, prolonged sleep, frequently interrupted by headache or pain in the limbs, possibly accompanied by tonic spasms. Later there are nausea, vomiting, abdominal pain, tenderness and dyspnea. In most cases a dense white mist before the eyes was complained of, and in many cases amaurosis became complete in from two to six days, lasting from a few days to a week. Ocular pain is infrequent. Color perception returns late, if at all, or incompletely. Death occurred in 8 cases within 24 hours, and in 5 within 48 hours. One man recovered after he had apparently reached a moribund stage. Thirteen necropsies gave similar results, namely, a post-mortem redness of the skin, less bright than in carbon dioxide poisoning; marked rigor mortis, affecting the erectores pilorum; muscles unnaturally red; blood cherry colored; lungs hyperemic and edematous, as were also the liver, spleen and kidneys; the vesical mucosa usually congested; usually marked hyperemia of brain and meninges. The treatment recommended is irrigation of the stomach, hypodermic injections of pilocarpine, vapor baths and packs, and copious warm packs.

JELLIFFE.

LOCALIZATION OF THE GREAT BRAIN: THE FRONTAL LOBE. C. von Monakow (Ergebnisse der Physiologie, III., 2, 1904).

The author sums up the present knowledge of this subject pretty thoroughly, going over the work of Flechsig, Broadbent, Hitzig, Bianchi and Ferrier. Broadbent distinguished between the sense areas of the cortex and those other cortical fields where the percept was elaborated into the concept, between the areas in which terminate the projection fibers from the subcortical portion of the brain and those without such fibers. Flechsig carried this further in his myelination studies, and made the distinction between projection and association centers; that is, between the primary centers for the representation of sensation and motion and the centers where these primary sensations were fused, to the latter of which he gives the name of "thought organ." His later researches, and those of other observers resulted in dividing the myelogenic fields into three groups, the first consisting of the projection center, early furnished with fibers, that is, the primary region; the second consisting of the association centers where the fibers appear later, that is, the terminal region, and the third an intermediary center. The second group is again divided into temporal and parietal, frontal and insular. If destruction occurs on both sides the intelligence is defective. The tempero-parietal centers deal with the recognition of spoken language and of the nature of objects, implying when united positive knowledge and imaginative capacity. The percepts of articulated language and those of hearing are united in the insula and in the prefrontal area are gathered all the impressions of conscious bodily life from which spring the acts of will. Hitzig, Bianchi and Ferrier consider the frontal lobe the organ of higher psychical activity, but our knowledge of localization even of the simpler operations is too slight as yet to justify much categorical assertion. To be sure, the area of frontal cortex in man is much larger in proportion than in the lower apes and beasts of prey, but on the other hand, in the horse and the goat it is nearly equal proportionately to that of man. Again, destruction, especially when bilateral, is often followed by changes in character and alienation of mind, and experimental removal in animals confirms this evidence; but on the other hand, bilateral frontal lesions have occurred without mental defect, and such defect has been associated with other lesions. Consequently, although the weight of evidence bears toward the special relation of the frontal cortex to psychical events, that fact cannot be said to be demonstrated. Indeed, we encounter difficulties in attempting to localize even the simplest mental operation, since the mere perceptive act implies components which are not physiologically defined or analyzed with sufficient fulness.

JELLIFFE.

NEURO-FIBRILS, AS DEMONSTRATED BY CAJAL. Azoulay (La Presse Médicale, January, 1905).

In this article Dr. Azoulay takes up the neuro-fibrils in leeches. The results of experiments with these lower forms of life support, as do those in the higher forms, the neurone theory of the nervous system. In the cells of the nerve ganglia the neuro-fibrils form one or two networks between the cell membrane and the nucleus. From this network both motor fibrils and fibrils of association pass out along the single cell process into the interior of the ganglion, sending off collaterals ending in the plexiform substance of the ganglia, as do also shorter processes from the cellular reticulum. The independence of the neurones is distinct, as in the higher animals, none of the fibrils anastomosing with each other outside the cells.

JELLIFFE.

Inequality of the Pupils in Pleuritic Effusion. Chaffard and Laederich (Arch. Gen. de Med., March 7, 1905).

In 7 of 17 cases observed by these authors the pupil was found dilated on the same side as the effusion. No dilatation could be detected in the other ten cases. In all cases it was slight and required careful looking for under favorable circumstances, accommodation being relaxed, as the pupils will be equal if strong light falls on the eye, or if the patient fixes his eyes on an object close at hand. The observations were made daily and the dilatation was found to vary, sometimes disappearing entirely and appearing again a few days later. In no case did it continue after absorption of the fluid was complete. The nature of the pleurisy seems to have nothing to do with the dilatation, as in both the negative and positive cases the effusion was due to various causes. It has been suggested that the dilatation results from compression of the branches of the sympathetic, but the fact that thoracocenesis has little or no effect on the dilatation tells against the idea of compression of nerve fibers by the collection of fluid. The authors suggest in this connection Schiff's law that all peripheral stimulation produces dilatation of the pupils, and refer the condition to the inflammation of the pleura. Jelliffe.

Subacute Combined Degeneration of the Spinal Cord. J. A. Ormerod (Bartholomew's Hospital Reports, Vol. XL).

The author reports a case of this disease, and notes the importance of recognizing it, since it runs a more rapid course than most other forms of spinal degeneration and usually results in death within a comparatively short time. It appears in middle age, and is often associated with anemia. Three stages can be distinguished, characterized in turn by ataxic paraplegia with paresthesia, by marked anesthesia with spastic paraplegia, and finally by flaccid paralysis with muscular atrophy and loss of the deep reflexes, total anesthesia, paralysis of sphincters, pyrexia, coma, and death in about six weeks, generally after the appearance of constitutional symptoms. The most characteristic features in the morbid anatomy of the cord are primary degeneration of the principal ascending and descending systems of fibers with a diffuse degeneration of the white matter in the dorsal region. The case recorded is that of a compositor, 57 years old, with no significant personal or family history. The first stage lasted in his case about a year, comprising failing memory and numbness in the fingers, numbness in the knees during the latter half of that time, and anemia after eight months. The second stage, nine months in duration, showed failure in ability to walk, then incontinence of urine, anesthesia in lower limbs, exaggerated deep reflexes and extensor plantar reflex. For several months thereafter no change occurred for the worse, and the legs even seemed better. Then the paraplegia became more pronounced, as did the anesthesia and mental deterioration. The third stage terminated in death within three days, having set in with rigor and pyrexia, vomiting and diarrhea, and passing into coma. Autopsy revealed degeneration of the ascending tracts of the posterior columns, of the ascending cerebellar tracts and of the pyramidal tracts. Jelliffe.

Juvenile Tabes. Hirtz and Lemaire (Rev. Neurologique, March, 1905).

The authors of this paper have gone over very thoroughly the existing literature of the subject, and arranged in tabular form the clinical data of 46 cases in patients between the ages of 5 and 22 years. The antecedent syphilis in these cases was, with a few exceptions acquired in the first months of life, hereditary. Urinary troubles, lightning pains or amblyopia are the manifestations of the trouble in early life, the first being the most common initial symptom, the second the next common, and amblyopia figuring more rarely in this capacity. In two cases noted the first symptom was gastric crises. It will be seen that the rule here differs from that in adults, in whom lightning pains appear as the initial symptom in about 62 per cent., whereas in children they occur in about 50 per cent. In all the juvenile cases incoördination was slight, and ataxia marked in only two of them. The authors consider the disease in children or adolescents as dependent on syphilis, and that this is at least probable even where no history of syphilis can be obtained. The authors are not prepared to assert that juvenile tabes implies necessarily a neuropathic organization as a concomitant of the specific toxin, but have noted sometimes in the family history of these patients epilepsy, tabes or general paralysis. They look upon tabes as an exceptional manifestation of the spinal affections resulting from hereditary syphilis, the diffuse form being more common.

JELLIFFE.

LEAD POISONING. Joseph Sailer and John M. Speese (Journal A. M. A., May 13).

The authors ex^m^ned the gastric contents, after test meals, in 12 cases of lead poisoning, and report the findings. The stomach contents were tested for lactic acid, free HCl and amount of pepsin present. In all cases a microscopic examination was made for the Oppler-Boas bacillus.. Their conclusions are as follows: 1. In a series of 12 cases of lead poisoning, or of suspected lead poisoning, deficiency in the secretion of HCl was noted in 10 of the chronic cases and was not observed in 2, one of which was doubtful and the other acute. 2. This deficiency in the secretion of free HCl in the majority of cases is associated with an extreme reduction in the percentage of peptic digestion, and with the presence of lactic acid. 3. It is not justifiable at present to regard it as an indication for treatment, at least not until the effects of the ordinary treatment for achylia gastrica in cases of lead poisoning have been tested. The authors have found no similar studies in the literature, which they consider rather remarkable in view of the pronounced gastric disturbances produced by lead poisoning.

AUTOPSY FINDINGS IN EPILEPSY. B. Onuf (Journal A. M. A., April 29).

Onuf reports the results of careful autopsies on 16 epileptics at the New York State Institution for Epileptics. In 12 cases there were valvular changes of the heart most frequently of the mitral valve (80 per cent.), less so of the aortic and still less frequently of the tricuspid valves. These he considers generally as secondary results of the special strain due to the major epileptic attacks. Capillary changes, tortuosity and aneurismal dilatations were observed in several cases, and were attributed to the same causes. In eight of the cases where the lungs were examined there was acute pneumonia as a contributory cause of death. The cerebral changes were very striking. In 10 cases there was a marked thickening of the pia, chiefly over the frontoparietal lobe. In other cases there were vascular lesions, circumscribed atrophy of one frontal lobe, subdural hemorrhage (one case), internal hydrocephalus (one case), cerebellar cyst (one case), and shrinkage of convolutions of vermis and adjoining cortex (three cases). The most striking changes, however, were noted in the thalamic region. These were in the nature of atrophy, sometimes the pulvinar, sometimes the other portions being most markedly affected. There was also an apparent discrepancy in the proportions of the geniculate bodies. Onuf discusses the possible relations of these thalamic changes to the epilepsy, but does not venture to express an opinion as to whether they are directly connected with the seizures or are only part of a general pathologic condition of the brain. He suggests that there was probably an optic atrophy in some of these cases, and hence the importance of fundus examination in epileptics. The imporeance of good clinical histories in these cases is also emphasized.

LARYNGEAL PARALYSIS. Hill Hastings (Journal A. M. A., June 3).

This is a report of a patient suffering from shortness of breath, pain in the chest, feverishness at night, loss of weight and hoarseness, but without cough or the physical signs of tuberculosis. Laryngeal examination revealed complete paralysis of the left vocal cord. This, with other physical signs, such as apex beat displacement, dulness and pulsation in the second intercostal space, and probable slight tracheal tugging, suggested the diagnosis of aortic aneurism pressing on the left laryngeal nerve, and this was confirmed by the X-ray, which revealed a pulsating shadow above the heart, extending one and one-half inches on each side of the sternum. The case is reported on account of its laryngologic interest as illustrating the value of an examination of the larynx in throwing light on the diagnosis. In this case the history rather pointed to tuberculosis of the lung with secondary laryngeal involvement.

JUVENILE APHASIA. C. H. Henninger (Journal A. M. A., June 3).

The causes of lack of articulate speech in 100 inmates of the Western Pennsylvania Institution for Feeble-Minded have been examined by the author. Thirty-five of these patients were epileptics, 14 of them also paralytics; 20 were cases of cerebral paralysis and 44 patients were idiots; 29 of the genetous type and 11 microcephalous, 2 hydrocephalic, 2 Mongolian and 1 cretin. Epileptic dementia from severe and frequent convulsions was credited as the cause of the aphasia in 6 of the epileptics, but in most of these cases he considers both the aphasia and the epilepsy alike due to some developmental defect and advises a careful search for mechanical impediments to speech. If the patients do not then respond to treatment an unfavorable prognosis should be given. In cases of cerebral paralysis attempt should be made to educate the uninjured hemisphere whenever there appears to be any possible chance of improvement. The head circumference in all the cases classed as microcephalic was less than seventeen inches, and aphasia was simply due to apraxia.

TETANUS. J. M. Anders and A. C. Morgan, Philadelphia (Journal A. M. A., July 29).

The authors give a preliminary report of their statistical study of 1,201 cases of tetanus, collected from the literature and by direct correspondence, with special reference to the incidence of the disease in the United States. They find convincing proof that tetanus is invariably the result of the introduction of the germ, and that the so-called rheumatic or idiopathic tetanus does not exist. They also find that it is endemic in all large centers of population, that in some localities where it was formerly common, notably in Long Island, it has become rare, and that occasional small epidemics, traceable to a definite source, occur in limited localities, as for instance, in hospitals, etc. It apperas also that tetanus is more prevalent in the hotter part of the year, that males are more subject to it than females, and that it is less frequent in advanced age. The robust are more susceptible than the weak, and the nervous than the lymphatic. There is much evidence that the disease is transmissible and may give rise to epidemics. The germ, Nicolaier's bacillus, is rarely introduced by the alimentary tract, but usually through open wounds, all parts of the body being very susceptible. A number of interesting clinical features observed in the cases collected are related, and it was noticed that the characteristic symptoms, especially trismus, were generally present. The diagnostic importance of the tonic contractions as opposed to the intermittent ones in certain other conditions that stimulate tetanus, such as strychnine poisoning, is emphasized. The authors found that their studies supported the earlier ones as regards the mortality, which decreases gradually after the tenth day and rapidly after the fifteenth. The study showed clearly the value of immediate radical local treatment, and that the most important thing is to open the wound freely in all directions under general anesthesia. Many patients were more or less benefited by the local carbolic acid treatment, and some observers report good results from the local use of ice or freezing mixtures or treatment in a cold room. For palliative treatment, chloral and the bromids appear to have been most extensively used. Calabar bean has been much employed, and also morphin, which should be used with caution on account of its inhibitory action on the respiratory centers. There is no question as to the value of antitoxin as a prophylactic; the tesetimony is uniformly in its favor. It should be used in any case in which there is suspicion of tetanus infection. In a well-developed case of the disease it has no appreciable beneficial effect, neither reducing the mortality nor hastening recovery.

EPILEPTIFORM ATTACKS COMPLICATED BY DOUBLE OVARIAN TUMOR. Russell (Glasgow Medical Journal, February, 1905).

The author reports the case of a woman, 31 years old, who had in October, 1899, a typical epileptic attack. About three years before that time she had had her first attack, and for a year had suffered a repetition of it at each menstrual period. Then for two years she was free from attacks. In November, 1899, she had two fits just before the menstrual period, although she had been dieted and put on bromides. In 1903 she had had but a few fits and was in good health. In May she had an acute attack diagnosed as peritonitis and abdominal tumor, torsion of the pedicle probably being the cause of the peritonitis. The tumor increased rapidly, and in July a large ovarian tumor was removed from the left side and a small tumor from the right ovary. She made an uneventful recovery, and has taken up her work as medical attendant without any recurrence of the epileptiform attacks.

PERIPHERAL NERVE SYPHILIS. J. Grinker, Chicago (Journal A. M .A., July 15).

The author reports the case of a man, aged 40, with history and traces of anterior syphilis suffering with right trigeminal neuralgia, right-sided peripheral facial paralysis and neuritis of left sciatic and left anterior crural nerves. The combination of nerve affections undoubtedly pointed to syphilis as their cause, though two months' vigorous antisyphilitic treatment failed to give relief. JELLIFFE.

A CONTRIBUTION TO THE PATHOLOGY OF ALCOHOLIC NEURITIS. Harlow Brooks (The Medical Brief, July, 1905).

The author, in a very interesting paper, reports the results of investigations, in connection with Dr. Lambert, upon several selected cases. Contrary to the very prevalent idea, the lesion is found to be central rather than peripheral. The ganglion cells of the cortex, particularly of motor area, exhibiting more or less marked degenerative change. The process may be traced downward through internal capsule and descending tracts of the cord. With the ganglion cells of the anterior horns, and isolated fibers of the anterior nerve roots, marked degenerative changes are apparent, as also of ganglion cells of the posterior root ganglia. Occasional fibers in the ascending tracts, especially of pronounced cases of this type of neuritis, exhibit degenerative processes. The peripheral nerve lesions, apparently, are secondary to those of cord and posterior root ganglia. The result of these investigations serves to confirm the earlier opinions regarding this disease, and offers a satisfactory explanation of the so-called "Korsakoff syndrome."

J. E. CLARKE (New York)..

A CASE OF MYOPATHY WITH MENTAL DEFICIENCY. Ernesto Tramonti (Gazetta degli Ospedali, May 14, 1905)..

The patient was a child who developed normally in every respect until as the result of some external or internal toxemia (gastro-intestinal?) and a bad heredity it presented certain symptoms of the nervous system partly motor, partly psychical. The disease took the form of a primary myopathy of the type of a pseudo-hypertrophic paralysis. There was muscular atrophy, a peculiar enlargement of the calves of the legs and gluteal region, absence of fibrillary tremor and reaction of degeneration, characteristic attitude, all of which point to this diagnosis. In regard to mentality we have had a progressive degeneration, and a normal child who has changed into an imbecile or something worse.. The mental condition has not developed all at once, but the changes have followed the physical changes in their appearance. For this reason we cannot regard them as simple complications, but rather as dependent on the same fundamental causes, affecting simultaneously both the motor system and the psychical centers. NOYES (New York).

PREVENTION OF APOPLEXY. C. Allbutt (Bristol Med. Chir. Jour., March, 1905).

Allbutt advises that every person over 40 should have his blood pressures measured every five years until he is 60, to the end that if a persistent mean rise of the arterial blood pressure is detected he may take steps to combat the tendency to apoplexy indicated thereby. If detected in its earlier stages, as would be the case in this procedure, this pressure may be controlled by regular exercise, abstinence from alcohol and reduction of the amount of food ingested. JELLIFFE.

ALTERNATING SINUSOIDAL CURRENT AND INTERRUPTED CURRENT IN THE CURE OF BASEDOW'S DISEASE. Giuseppe Severino (Gazetta degli Ospedali, May 14, 1905).

The writer concludes that in four patients suffering from Basedow's disease who were treated by this electrotherapeutic method satisfactory results were obtained, more or less improvement occurred, and the more troublesome symptoms were controlled. The electrical current was applied over the cervical sympathetic, and a mild interrupted current produced the best results. If the sinusoidal current is used it should be applied directly to the thyroid gland. NOYES (New York).

THE CURABILITY OF TABES. Faure (Gaz. des. Hop., December, 1904).

The author discusses the contradictory opinions as to the curability of tabes and the mercurial treatment, accounting for their divergence by the following facts: There is a wide variety among cases of the disease itself. Analogous cases treated by different methods of administering mercury will show different results. The patients vary largely as to toleration of mercury, the younger ones in the earlier stages of the disease supporting it best. The care exercised in directing the treatmnt is an important factor, as unwise management may occasion an aggravation of the disease when proper care would enable the patient to attend, in a modified manner and with frequent rests, to his business. The dose must be adjusted to the patient's strength. Some lesions are unaffected by mercury, and some cases get worse in spite of the most careful treatment. The author thinks that inunctions and the injection of insoluble salts is uncertain, and advocates the use of soluble salts. He also emphasizes the fact that rest and general hygiene are of the utmost importance.

JELLIFFE.

Book Reviews

Grundlinien einer Psychologie der Hysterie. Von Willie Hellpach, M.D., Ph.D., Nervenarzt in Karlshrue. Wilhelm Englemann, Leipzig.

Hysteria is in the air. No less than five or six large monographs dealing with its various phases have appeared within the last few years. Whereas since the days of Brissaud and Charcot the hysterical field has been almost exclusively cultivated by French savants, there has been of late years a great activity on the part of German investigators, who in large part are rediscovering many of the manifestations already made known by their Gallic confreres. Hellpach has given us an extremely valuable work. Whereas from the literary side it lacks the ease of diction and the clearness of description that characterize Janet's work, nevertheless the student of hysteria will find much not already covered by Charcot, Janet, Vogt, Binswanger, Freud or Löwenfeld, to the last of whom Hellpach really owes his initiative.

In the opening chapters he discusses the Problem of Hysteria, reviewing in brief the developmental history of the disease. To our mind he is supercilious in his bestowal of praise upon the early French workers. He is particularly hard on Sollier for his mechanical explanations, saying that the bad habit of anatomical psychology is so deeply rooted as to form an actual impediment to progress. We anticipate when we express the fear that too wide a deflection from conceptions based on anatomically localized ideas is apt to throw us into the worse slough of metaphysics which has truly been the ignis fatuus of all psychological discussion. We confess we prefer the reaching out after the anatomical conceptions of Flechsig, Wernicke and others to the metaphysics of many of the German authors. Other portions of this same chapter deal with hysteria as a disease process, and with logical considerations of psychopathology.

The second portion of the volume deals with the Analysis of the Hysterical character. The author makes much of suggestion. placing it in the front rank as a psychical factor, then describes the hysterical movement anomalies, the modifications of sensation, the intellectual changes and the psychophysical limits to the psychology of hysteria.

These chapters are full of suggestion. We cannot escape the conviction that there is a tendency to spin the fine figment of descriptive phraseology to its extremest filmiest limits, at times almost incomprehensible to the unsaturated metaphysician, but throughout the author rests his arguments on sound precepts.

The closing chapters, on the Genesis of Hysteria, offer the reviewer the greatest satisfaction. In the discussion of the child nature and its relations to the hysterical concept we find our author at his best in most common sense and understandable phrase. In the *Lenksamkeit*, the ease of guidance, the suggestibility of the childish nature, the author sees the source of hysteria, and in the preceding chapters had made a thorough analysis of this state of guidability.

Taken as a whole, Hellpach's contribution is one worthy to stand with the masters in the elucidation of the problems of hysteria.

Jelliffe.

PUBLICATIONS ON THE SUBJECT OF MILITARY SANITATION. Issued by the Medical Division of the Prussian Royal War Department. Number 29. Studies from the Laboratories of Chemical Hygiene, Part I, Berlin, 1905.

A collection of seven papers on the age limit of mustard plasters and calomel tablets, the quantitative determination of iodine, the rôle of fatty acids in fat-impregnated clothing, the determination of caoutchouc, the usefulness of legumen-meal, the significance of animal and vegetable extracts in nutrition. NICHOLS (Washington).

TEXT-BOOK OF NERVOUS DISEASES AND PSYCHIATRY. By CHARLES L. DANA, A.M., M.D., Professor of Nervous Diseases in Cornell University Medical College, Visiting Physician to Bellevue Hospital, etc. Sixth Revised and Enlarged Edition. William Wood and Co., New York.

To have been able to prepare a work on nervous diseases which has appeared in six editions is no mean achievement. The stamp of general approval has been placed on Dana's text-book, and we cannot feel that we can add much to that which has already been said about his work.

No notable changes have come about in this present edition; it remains much as we have known it, a concise, descriptive work on nervous diseases, of great value to student and practitioner alike. The minor criticism that certain points of view have not been changed, as for instance the statement regarding the sensory nerves that they go to their points of origin instead of coming from them, is of little moment when one considers the work as a whole. The author tells us that "the optic nerve *originates* in the *retina*, just as the olfactory nerve *arises* in the peripheral cells of the *olfactory mucous membrane*," p. 162, and yet, p. 159 says that the olfactory nerve *arises* from the *olfactory* bulb and is distributed *to* the mucus membranes." "The two parts of the auditory nerve," p. 86, "pass *to* the cochlea and semicircular canals." Why not originate in the ganglion cells there, as the analagous nerves, the optic and the olfactory?

We wish the author would clear up the differences between chronic anterior poliomyelitis and progressive muscular atrophy. Is the difference purely "artificial," as Oppenheimer states, or are there two separate entities here to be considered?

The new contribution that Dana makes in this revision is a short section on mental diseases. There are 68 pages full of meat. The author adopts the "adaptation to environment" definition made so popular by Maudsley and Mercier, and certainly no better point of view of insanity has been put forth. The author's adaptation of the contentions of Charcot, Brissaud, Janet and their followers, regarding the psychic nature of many of the neuroses, so-called, is instructive. The chapters on Neurasthenia and Hysteria do not show the point of view quite so clearly.

We are not in accord with the author's use of the word Functional and Organic Insanities, if he means thereby that there are no organic lesions for Dementia Precox while there are in General Paralysis. Because the defective fiber tracts in paranoid brains have not been pointed out, we do not think it justifiable to perpetuate the old word "functional" in psychiatry after it has worked so much havoc in neurology and led to confusion rather than to clearness of thought.

The chapter on general psychology is an admirable one for conciseness, and that on general symptomatology very well put. Dr. Dana's polypathic insane man, although extremely sketchy, is admirable, particularly for students.

For clear, short descriptions of mental variations the systematic portion of this section is highly commendable. They are of particular value as coming from a gifted observer who has studied his patients for the most part outside of asylums. We can only add that we regret that they are so didactic in view of the extreme variability in the domain of mental manifestations. We welcome the sixth edition as we have the former ones.

JELLIFFE.

THE DOCTOR'S WINDOW. Edited by Ina Russelle Warren. THE DOCTOR'S RECREATION SERIES, VOL. V. Edited by Charles Wells Moulton. The Saalfield Publishing Co., Chicago, New York and Akron, O.

This latest volume of a cheerful series embodies an exhaustive collection of "poems by the doctor, for the doctor and about the doctor," and takes its title from the opening selection by Austin Dobson. The entire field of English literature has been ransacked to fill these pages, and from Chaucer to Henley a rich harvest of song has been gathered. It is hard to say which gives the more pleasure, to find old favorites included here, like Henley's "Romance," Dr. Holmes' "Morning Visit" and the sportive quatrain of "I Lettsom," or to discover new and entertaining verse by some author unheard of before. Perhaps the chief occasion for surprise is the number of poems which can be classified under the first item of the subtitle, poems *by* the doctor. Dr. Holmes, Dr. S. Weir Mitchell and Dr. Andrew H. Smith we expected to find here, and are not disappointed but here are an innumerable throng of other M.D.'s handling rhyme and rhythm with a grace and gusto that seem to imply some essential relation between the qualities of poet and physician. Sportive, serious or sentimental, the verses offer something for every taste. Among the poems about the doctor one cannot help noting the proportion devoted to eulogies in one form or another of the "Country Doctor," and certainly if any particular class of the profession is to be singled out for special attention, none deserves it better than "The grim old country doctor, the kind old country doctor," who too often "works for fun and boards himself."

GOODALE.

PSYCHIATRY: A TEXT-BOOK FOR STUDENTS AND PHYSICIANS. By Stewart Paton, M.D., Associate in Psychiatry, the Johns Hopkins University, Baltimore; Director of the Laboratory, the Sheppard and Enoch Pratt Hospital, Towson, Maryland. Lippincott Company, 1905.

Stewart Paton is, in the psychiatric realm, one of the children of light. The series of complex stimuli (with apologies to Dr. Paton for our style) derived from sources which we commonly refer to as "Johns Hopkins" and "the Pratt," impinging upon a highly organized cortex, would naturally culminate in a reaction in the form of a scientific book like the one before us.

It is a good book. By reading it any one may make up, who has failed to keep track of the recent writings on insanity. As a digest of the literature, it has no equal in any language.

Of the first eight chapters, devoted to principles, the best, perhaps, are on the "Modern Hospital for the Insane," and, in part, the "General Causes of Insanity," the latter containing the soundest discussion of heredity that we have met with. In the remaining chapters, IX to XXI, the forms of insanity are taken up in groups, as the "Manic-depressive Group," "Dementia Paralytica Group," etc.

This kind of title betrays an abhorrence of "entities," expressed on page 181 thus: "Epilepsy, which may very properly be taken as a prototype of alienation, is *not in any sense* an entity, but a condition or symptom-complex, . . . and the same is true in regard to all the various psychoses," etc.

That is perfectly scientific, as drawn from minute study, but pushed to the extreme in the discussion of the "Paranoia Group," it becomes the fault satirized in Hudibras:

"By geometric scale
To take the size of pots of ale."

As this is not altogether for neurologists, but "a text-book for students and physicians," the reader should not be set down in the midst of bewildering psychologic uncertainties; he should be lead as a child to see first, as the epistle says, "that which was from the beginning which we have heard, which we have seen with our eyes"; that is, less figuratively, by Herbert Spencer's principle of developing an idea in the order of its historic development, telling first what Esquirol and Morel meant by that which later was called paranoia.

Supose, in some practice, the opening gun of a chapter on typhoid fever spoke of pneumonia of typhoid origin, of various forms of typhoid neuritis, and bone-caries, of bacillus-coli infection and para-typhoid, of certain confusing grippe and tuberculosis cases, and of typhoid fever without fever, what agnosticism would it engender!

Suppose, on the other hand, typical paranoia were found to have a bacterial cause (which, of course, is incredible), still, the transitions into other phychoses would vex us.

Paton's brief chapter on paranoia begins in scepticism and ends in agnosticism.

This, doubtless, is a fault of erudition. Beyond this we have only praise for Dr. Paton's book. It is broad-minded and judicial, thorough and correct. It is a concordance of the clinical knowledge, pathology, psychology, therapeutics and sociology of insanity—a complete psychiatry.

And there is something heroic in the high stand it takes for science at every point.

WILLIAM PICKETT.

News and Notes

A NEW VIEW OF THE ETIOLOGY OF INSANITY.—The gallant President of the National Antivaccination League, writes the *British Medical Journal* of Sept. 23, was in good form at a guardian's meeting last week. The subject under discussion was the great increase in lunacy. Some people talk of love as leading to insanity, and others blame alcohol. But General Phelps, as reported in the *Birmingham Evening Dispatch,* knows better, and those who know him can have no difficulty in knowing what he thinks he knows. He declared that "neither drunkenness nor love affairs were causing more insanity, and he thought that they would "find that the one cause of the increase was vaccination." Of course it was, and it appears that all the King's Norton Guardians but himself must have been vaccinated, for he evidently regards them all as madmen. He cannot hope for their permanent recovery, but he really would like that they might be blessed with just a brief period of temporary sanity to enable them to see the real truth of the vaccination question. A mere glimpse would be enough. "If ever they had a sane moment, they would find that vaccination produced small-pox, promoted cancer, and resulted in a great increase in insanity." It is seldom we agree with the President of the Antivaccination League, but we are bound to admit that there is a sense in which vaccination is related to lunacy and lunatics. If there were no vaccination there would be no antivaccination, and no Antivaccination League, and no President of the League. In these quiet times, when Parliament is not sitting and the supply of nonsense is consequently at so low an ebb, it is very good of General Phelps to step into the imminent deadly breach, and he deserves the thanks of all of us for his very pleasant little contribution to the national gaiety.

BELGIAN CONGRESS OF NEUROLOGY AND PSYCHIATRY.—The first Belgian Congress of Neurology and Psychiatry was held at Liege on Sept. 28, 29 and 30, under the patronage of the Government and the Honorary Presidency of the Ministers of Justice and Agriculture. Among the questions to be discussed were the following: (1) Work considered as a therapeutic agent; (2) lumbar puncture from the diagnostic and therapeutic points of view; (3) the sense of pain. Visits were paid to the principal university establishments, and to the lunatic asylums of the district.

PRIZE COMPETITION FOR PAPERS IN PREVENTION OF LEAD POISONING.—The International Association for Labor Legislation is prepared to offer a prize of £250 for the best treatise on the prevention of lead poisoning in mining and milling lead ores; £500 for the best treatise on the prevention of lead poisoning in smelting and refining works; two prizes, £125 and £75, for similar treatises relating to white-lead works, paint works, manufacture of electrical accumulators, etc.; 4 prizes, (1) £75, (2) £50, (3 and 4) £37 10s. each, for house, ship and coach painting, etc.; and four prizes ranging from £75 to £37 10s. for the best treatise on the prevention of lead--poisoning in trades where raw and manufactured lead are consumed or handled, as in type foundries and printing offices. The essays must be sent in to the International Association for Labor Legislation, Basle, Switzerland, not later than May 15, 1906. Leaflets giving full details can be had by applying to the Honorary Secretary, British Association for Labor Legislation, Club Union Buildings, Clerkenwell Road, London, E. C.

In the death of Dr. Carl Wernicke, Professor of Psychiatry at the University of Halle, which took place on the 15th of June, following a bicycle accident, psychiatry has lost one of its most original thinkers. Dr. Wernicke's "Grundriss der Psychiatrie" has been one of the most stimulating volumes, and his researches on aphasia have promised more to elucidate some of the problems of psychiatry than those of almost any other writer. He has been constant in his study of the problems of aphasia since 1874, when as a young man of 26 years he wrote on the aphasic symptom-complex. Three of his pupils, Gaupp, Heilbronner and Bonhoeffer, are now professors in important psychiatric chairs, and his influence has been widespread in psychiatric circles. One of his most recent contributions to the subject of aphasia has been discussed in the *Deutsche Klinik*, and a very able résumé has been made by Dr. Adolf Meyer in the *Psychological Bulletin* for Aug. 15.

Wernicke's chief contributions have always had a distinctly localizing tendency, and all those who have been stimulated by his writings have fallen under the sway of his method, which has called for great clearness of concept. Wernicke always sought to establish an anatomical substratum for his work, and never strayed into metaphysical fields.

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THE
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Original Articles

A STUDY OF DEMENTIA PRÆCOX.*

BY D'ORSAY HECHT, M.D.,
OF CHICAGO,
INSTRUCTOR IN NEUROLOGY AND CHIEF OF THE NEUROLOGIC CLINIC, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL; ASSOCIATE ATTENDING NEUROLOGIST TO THE COOK COUNTY INSTITUTIONS AT DUNNING, ILL.

Mindful of the fact that this paper is intended for those conversant with latter day problems in psychiatry, I shall studiously avoid lengthy reference to the general problems more or less intricately interwoven with the age of adolescence. Rather shall I assume that the psychologic phenomena, operating with especial force at the period of pubescence, are well understood, and that the newer qualities of mind, body and soul ushered in with and incident to a subsequently unstable, formative period of life, require no further elucidation. With this due acknowledgment then of a psychophysical adjustment capable of making or marring the individual in the body social, I shall attempt a review of some of the psychiatric literature of several decades past, chiefly for the purpose of emphasizing the major evolutional phases through which the chapter on dementia præcox has passed.

The first reference to a mental state that has something remotely in common with dementia præcox is to be found in a small highly interesting volume entitled "Lehrbuch der Störungen des Seelenlebens," written in 2 parts and published 1818 by Dr. C. A. Heinroth,[1] then Professor der psychischen Heilkunde in Leipzig. On pages 347-351 of Volume 2 of this work, the author treats of "Die Willenlosigkeit" in its 4 forms, viz.: (1) Die reine Willenlosigkeit (abulia simplex); (2) Willenlosigkeit mit Schwermuth (abulia melancholica); (3) Willenlosigkeit mit Blödsinn (abulia anoa); (4) Willenlosigkeit mit allgemeiner psychischer depression (albulia catholica). Of the "abulia simplex" which alone concerns us, Heinroth says, "Der Wille des Kranken ist gebunden; der Kranke ist in Beziehung auf den Willen unfrei," meaning substantially that the will of the patient is bound down. Under the heading of semiotische, diagnostiche, prognostiche Momente he adds: "The whole appearance, the posture, the attitudes of the patient bear the stamp of his condition. His glance is lifeless—the face expressionless except for a dulness, which is the result of inactivity. His posture is one of languor and inattention. His movements slow and dilatory, etc." Reading the entire text of about 2 small pages in all, one concludes that the author may have been exploiting "melancholia attonita," and yet just suspicion is aroused that many points in the description bear close resemblance to the now more familiar symptom complex of dementia præcox.

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* Read at the meeting of the Chicago Neurological Society, May, 1905.

E. Esquirol[2] in his "Des Maladies Mentales" (Bruxelles, 1838, Tome 11, p. 65), discussed the several forms of dementia, but the severer cases described by him would to-day be classed with the katatonias. He also afforded ample proof of the recognition of the milder types as well, and called attention to the fact that the normal parallelism between psychic and physic forces existing in childhood is not always maintained. When the higher and superior faculties of the adolescent, says Esquirol, come to a standstill, and mental expansion ceases to keep pace with bodily growth, then we meet with the condition of accidental or acquired idiocy.

The literature for some twenty years (1838-1858) following this treatise is singularly devoid of all mention of dementia, and it is reasonable to assume that the chapters on hereditary neuroses, moral insanity and degenerative psychoses embraced all the distinctions previously accredited to dementia as an entity.

Beginning in 1857 with Rousseau's[3] thesis "De la folie a l'époque de la puberté," there appeared in rapid succession (Moreau, 1859),[4] (Morel, 1860),[5] (Maudsley, 1870),[6] (Legrand du Saulle, "Folie Hereditaire," 1873),[7] (Falret, 1876),[8] (Legrain, 1886),[9] (Langdon Down, 1887),[10] many contributions under the caption of hereditary insanity, depicting forms of simple arrested development (einfache Entwicklungshemmungen) wherein all intellectual function seemed brought to a standstill in hitherto mentally normal individuals and even those precociously endowed. The purpose of these essays was to lend scientific color to facts already well known, and but only partially understood by active educators of the young. They were intended to emphasize the mode of onset, the great tendency on the part of some of the brightest, most promising pupils of somewhat advanced age to go backward, ever backward, relinquishing all hold on previously erected standards, and sinking to the intellectual level of boyhood days, with its unmannerly and impertinent prerogatives.

The clinical knowledge of this early occurring insanity was ably reinforced by the observations of Tuke[11] in 1879 and Fink[12] in 1881. Tuke, with all the weight of his authority, insisted that in these disease-pictures more significance be attached to the unmistakably progressive character of the mental deterioration. He stated, "Puberty is often a causal factor in psychic disturbances, but patients so afflicted rarely ever become asylum inmates, in fact are seldom looked upon as insane. Exactly as in moral insanity, they are given to moral perverseness, dangerously impulsive acts, but present in addition undoubted progressive mental weakness." Mairet,[13] in 1888, published a treatise entitled "Folie de la puberté," in which he, even more than Tuke, gave prominence to the "demence."

Clouston[14] (*Journal of Mental Science,* October, 1888), in a presidential address (delivered at the College of Psysicians in Edinburgh, August 6, 1888), which has been voted a classic by all familiar with it, voiced for the first time the conviction based entirely upon clinical experience that amongst adolescents "there are many cases where previous excitement (he had in a preceding paragraph been speaking of dementia secondary to mania and melancholia) was so slight, so short-lived, that we must conclude that the essential nature of the mental disease was a tendency to dementia from the very beginning." Clouston by reason of a vast experience, and his rank as a profound thinker and classic writer, merits more extensive quotation. In one brief sentence, he summed up the mental phase of a large number of essential (idiopathic) dementias:—"The patients simply became less acute in emotion and judgment, less powerful in volition, less able to do their work or take care of themselves, and less social and more 'silly,' these symptoms gradually going to marked dementia." "Such cases," said Clouston, "have made a great impression on me and influenced powerfully my views of the real significance and causes of dementia." He then recites the case of a girl of 19 whose maternal cousin was insane and speaks of her as follows: "She was a well nourished, intelligent, cheerful girl, and who a year and half ago after hard work, in nursing, and the shock of her patient's death, as well as perhaps a love disappointment, began 'to feel as if something was coming over her,' as if she were 'losing her reason.' Then she showed less spontaneity, less cheerfulness, and spoke less frequently. Next she lost all interest in everything; would not get out of bed except by force, lost all sense of shame and modesty, would not occupy herself and had to be sent to the asylum, where she is now gradually becoming demented. She never was melancholy nor maniacal; there was no cortical storm at all. Such," adds Clouston, "is a case of true primary dementia." "The cases of so-called primary dementia of the authors are mostly cases of stupor, and the term should not be used any longer to distinguish them. We must reserve the term Dementia for incurable conditions of mental enfeeblement."

Prior to the year 1871, the German psychiatric literature is barren of any and all references, calculated to promote interest in or understanding of the adolescent insanities. Dr. Edward Hecker's contribution in Virchow's Archives in 1871,[15] on Hebephrenie ("Ein Beitrag zur klinischen Psychiatrie") was the first attempt at bringing order out of chaos. To-day discussion of dementia præcox is incomplete without due regard for this article of monographic thoroughness. Hecker at the outset alludes to England's intense interest and devotion to the problem of child-life insanities, including the conditions of idiocy and cretinism. He had been disappointed at the sparseness of the literature on diseases incident to the years of pubescence, all the more so because the types of mental alienation incident to this period were characterized by such distinguishing features. Some of the psychic disturbances at this age, are to be regarded merely as early exhibitions of forms of insanity, occurring in any of the latter stages of life, but said Hecker, "von allen diesen aber hebt sich durch eine ganz eigenthumliche Verlaufsart und ein Reihe besondere Symptome, jene Form der Hebephrenia ab." (Of all these forms that of hebephrenia merits distinction by reason of a series of special symptoms and a peculiar course.)

After disposing of several case-histories, recorded in a most accurate and painstaking manner, he correlated the most important features of hebephrenia, thus—"The onset in close succession of puberty; the appearance, alternately of melancholiac, maniacal and confusional states; a speedy psychic decline (psychischen Schwachezustand), with its finality in a terminal dementia, which may be anticipated from the very first."

Following this paper, there was a dearth of reports until Kahlbaum[16] undertook (*Allgemeine Zeitschrift f. Psych.*, Bd. 46, S. 46) to advance the problem under the guise of a new title, "Heboidophrenie." In an introductory passage, he claims the distinction of presenting a symptom-complex entirely novel. I quote: "Unter Heboidophrenie verstete ich einen psychischen Krankheitszustand, der in der Jugend auftritt und in seinen symptomatischen Eigenthümlichkeiten so geartet ist, dass er sich mit keiner anderen der bisher aufgestellten psychischen Krankheitsformen deckt." That Kahlbaum drew generously on the then prevailing hebephrenic type in constructing his own clinical picture is not denied, but if on the other hand we are to believe Kahlbaum's insinuation that Hecker's description in 1871 was wholly appropriated from material studied and convictions expressed by him, the original intimation of plagiarism may be discredited. Some of the chief conclusions arrived at in Kahlbaum's paper are expressed in short sentences as follows: (1) There does exist a psychic disturbance of adolescence, which does not admit of being grouped with the other known mental diseases. (2) This psychosis is distinguished by a radical change in the individual, by a complete alteration of character, temperament, and appearance. Kahlbaum desired to have it understood that in his

"Heboid forms" there was no initial phase of melancholia or mania; that these patients remained rather unattuned to their surroundings in a mild state of ill humor. He stated that the lack of insane ideas and absence of any dementia further differentiated these patients from the hebephrenias of the Hecker type and sought to introduce hebephrenia vera as the classic form terminating in dementia and heboidophrenia as a milder, abortive, curable form. After carefully considering some of Kahlbaum's citations, I conclude that with his Heboidophrenia he sought to establish a disease picture occurring in adolescence, in which neither maniacal nor melancholiac tendencies appeared, in which the more pronouncedly bizarre attitudes were absent, in which a peculiar mental weakness obtained that could well be ranged with the terminal mental states of the hebephrenic forms although of distinctly milder degree. In confirmation of the existence of an heboidophrenic group (à la Kahlbaum), we have Weygandt's[17] description of cases (Atlas und Grundriss der Psychiatrie") entitled "Dementia Simplex oder Heboidophrenia." The transitory hallucinations, the mannerisms, the rapidly progressive though mild form of dementia would make one prone to class these cases with those of true hebephrenia. That Kahlbaum and Hecker labored with better success in the vast and complex field of demented cases than their predecessors, is readily attested by their respective departures from an old classification or no classification at all, to the establishment of two distinctive types, the hebephrenic and katatonic.

Since this epoch-making advance many authors have entered the lists with observations and deductions, which if admittedly less radical have nevertheless materially enriched the literature. With some regard for their chronological order, the writings of Gauthier (1883), Cl. Neisser[18] (1887), Koch,[19] (1889), Charpentier (1890), A. Pick[20] (1891), Daraszkiewicz,[21] (1892), Griesinger[22] (1892), Sommer[23] 1894), Krafft-Ebing[24] and Wernicke[25] (1900), Bourneville[25] (1901), Diem[26] (1903), merit most creditable mention. In this connection, it is purposely intended to reserve for last but not least mention, the name of one other author.

Any endeavor to record in the medical literature something of psychiatric interest at once conjures up the name and work of that most distinguished of contemporary alienists, Emil Kraepelin, prior to whose activity, students in this field of work found themselves seriously handicapped with views erroneously held, concerning certain aspects of mental diseases and hopelessly involved in the meshes of a disordered clinical classification. To quote a paragraph from the remarks of Dr. Edward Cowles[27] (McLean Hospital, Waverly, Mass.) on "The progress in the clinical study of psychiatry," made before the Am. Psych. Assoc. at New York May 24, 1899—"the latest and most original contribution is that of Kraepelin, who in his studies and clinical methods is giving us perhaps the most illuminating conceptions of insanity that we have yet received as explaining principles."

Laboring in an atmosphere heavy laden with controversy, inadequate definition and misapplied terminology, it proved no easy task to present a new classification of psychoses, fashioned from logical premises and principles entirely novel to those steeped in the alienistic lore of an Esquirol, Moreau, Griesinger, Kahlbaum, and others of their kind and age. In his splendid endeavor to build on a solid and lasting foundation Kraepelin has been assisted by an unfailing adherence to the tenets of simplicity. This fact cannot fail of recognition when we note how with one sweep he correlates maniacal outbursts, mental depressions bordering on stupor and catalepsy, bizarre attitudes, successively appearing delusions and hallucinations, and subordinates them as a "series of disease pictures" to one feature which all have in common, namely, a termination in a special kind of mental weakness, which he is pleased to denote as "dementia præcox." Just how favorably this bolder design compares with the antique pattern will be touched upon later in the critical résumé. For the present, let me reflect only the American view as expressed by Dr. Adolph Meyer,[28] who in a brilliant exhaustive critique of recent problems of psychiatry (Church and Petersen), states, "This elevation of the 'outcome' to a nosological criterion is a very radical step. *A priori*, many objections in general medical principles revolt against it; we are far less concerned about the reason of the fact than about its certainty and possibility of predicting it from conditions which do not already include it, or include it so that it was not formerly recognized."

It is difficult to set down under exact dates the gradual advances made by Kraepelin[19] in this particular domain of psychiatry.

In the fourth edition of his Psychiatry, he writes a chapter on "Die psychischen Entartungsprocesse" (the degenerative psychoses) and arranges them in three groups: (a) Dementia Præcox (Hecker's Hebephrenia); (b) Katatonia (Kahlbaum's); (c) Dementia Paranoides. In the fifth edition, this classification is held to, but in the sixth edition (1897), the radical change is recorded for the first time, and three discrete clinical types are developed and exhaustively treated as but different expressions of one affection. As to the rationale of this change and the controversies that have grown out of it, more anon; for the present suffice it to say that students of modern psychiatry who value above all else the "summa summarum," owe their debt of greatest gratitude to Kraepelin more than to any other one author for this admirable scheme of generalization.

Definition and General Symptomatology.—This form of insanity has been inflicted with so many and diverse appellations that even the close observer will occasionally pause to identify it by title alone. It is the adolescent insanity of the English, the demenza primitiva of the Italians, the Jugendirresein of the Germans, the demence precoce of the French, the primary dementia of the Americans. That Morel is responsible for the designation demence precoce is I believe universally conceded, although some recent writers seem to think that the term is entirely original with Arnold Pick. In 1891, Pick,[20] in the *Prager Medicinische Wochenschrift,* describes two cases, and gives a few historic data under the following caption: "Uber primäre chronische demenz (sogenannte dementia præcox) im jugendlichen Alter." From this it seems clear, and Dercum[80] (*Journal Am. Med. Assoc.,* Feb. 4, 1905)has so stated it, that Pick of Prague first employed the Latin equivalent, translating it from Morel's demence precoce.

The almost total absence of the term dementia præcox from American and English text-books and to a great extent current literature is explained in part by this confusing provincial terminology.

The delinquency in the matter of an international or universal adoption of the name dementia præcox has arisen also from a speculative attitude towards its permanency as a clinical entity.

It is not possible to render a definition of dementia præcox that is at once terse and comprehensive. The symptoms are so notably diverse as even to preclude the possibility of a good perspective, except perhaps in the terminal stages when as Kraepelin states "the more accidental and transitory secondary symptoms are supplanted by the permanent and distinguishing changes in the mental life."

Dunton[81] (*American Journal of Insanity,* Vol. 59) very aptly quotes Kraepelin with regard to the scope of the disease as follows: "Judged by their termination a large number of cases of mental alienation fall into one or two groups. Those ending in recovery being regarded as instances of manic-depressive insanity, while those culminating in dementia represent some form of dementia præcox;" and he adds, "the initial symptoms of the two groups are sometimes so similar that a differential diagnosis becomes very difficult."

At the onset of his chapter on dementia præcox, Kraepelin does not indulge the reader in a long, paranthetically phrased, involved definition. Where his conception is broad, his conclusion is brief. He says, "Under the term dementia præcox we range a series of disease-pictures whose common characteristic is a termination in a peculiar weakened state of the mind. This unfavorable outcome need not occur in every instance, but is nevertheless so exceedingly frequent as to justify our holding fast to this general name."

Before going into clinical refinements, it might be well to discuss a few of the fundamental, basic symptoms, occurring at one time or another in all cases, and obtruding especially in the terminal stages, but be it understood that the remarks on the symptomatology of this disease are desultory and intentionally fragmentary. Since asylum alienists are privileged to observe and study their cases for indefinite periods and under the best auspices, it is from their full reports alone that the final word should be framed. Even the majority of psychiatric clinicians do not see their material long enough to enable more than premature reflection. To attempt more than generalization here would be presumptuous and therefore undesirable.

Most impressive in nearly all cases, is the remarkable dissociation between memory and judgment. Wherever the mentality remained sufficiently intact to apply tests along this line, most striking evidences of this disparity were noted. The case of C. H. (Case 1), seen at the Northern Hospital for the Insane at Elgin, Ill., is illustrative. The patient, a male, knew not the why or wherefore of anything he did. He could give no reason for being under restraint, and was more unable than disinclined to discuss any of his acts with even a glimmer of intelligence. The mental powers concerned in the formation of a decision were too sluggish to be of the least service to him in an estimation of self or surroundings. The machinery of judgment was surely out of commission. His memory, in no wise as greatly impaired as his judgment, was none the less defective. This young man was a musician. With him music was a study; he cultivated it as a vocation, not a mere accomplishment. To develop it, he gave most of his time and certainly his best mental powers. This school-gotten knowledge remained with him—it was assertive even in his state of mental bankruptcy to a degree of recollecting opus numbers of musical works and correctly naming sonatas in their major or minor keys. Remote events were remembered better than recent ones.

In the vast majority of cases, adult patients will know the year of their birth, the date of their graduation from the common schools, recall Sunday school incidents, but fail to remember when they came under institutional care—what day it is—how old they are or (in girls) when the last period occurred. During stuporous periods or under stress of delusions and hallucinations, even the best retentive memory will for the time being succumb.

The power of judgment (Urtheilsfähigkeit) is early and very materially impaired. These individuals may retain a full appreciation of old accustomed routine, but cease to comprehend the simplest innovations and fail to grasp the easiest situations; they are unable to understand what is going on about them; can neither reason nor reflect. Patients occasionally take slight cognizance of this mental deterioration; they seldom if ever appreciate the gravity of the trouble or understand the blight that is on them.

In conversation with a well-defined yet mild case, the train of thought (Gedankengang) is not maintained. I recall especially in R. D. (a male patient) an irresistible tendency to wander away from the topic of conversation (Sprunghaftigkeit) despite every effort to keep him keyed to the line of inquiry. In graver cases, this irrelevancy and interruption of ideas gives rise to exhibitions of confused speech (Sprachverwirrtheit) amounting not infrequently to complete incoherence. Sentences are loosely put together.

Whereas the comprehension of an idea may be quite correct, the expression of that idea is frequently loose and disjointed. Closely allied to such speech derangement is another anomalous form of expression,—verbigeration, a sort of senseless repetition of words, phrases or whole sentences. A few thoughts may completely possess the defunct mind and be obtruded automatically as it were for weeks and months through the mediation of speech in constantly recurring meaningless phrases. E. G.'s attitude in conversation was nicely illustrative of verbigeration. I recall her oft-repeated "go home—go home—shall go home, when I go home," in answer to various questions.

Within the past weeks (February 10) a boy of seventeen was brought by his mother to the Northwestern University Medical Dispensary. The train of symptoms stamped it clearly as an incipient case of dementia præcox. In answer to every question he repeated his reply two or three times. What is your name? Wm. F.— Wm. F.— Wm. F.—. Where do you live? 800 6th St.— 800 6th St.— How old are you? 17—17 years—17, etc.

It is Kraepelin's opinion that the conception (Auffassung) or perception of external perceptions is but little involved in dementia præcox, and in direct ratio the sense of orientation remains undisturbed. The patients know they are in asylums; know the physicians who visit them; distinguish their various callers; recognize fellow patients, and note changes in the dress of the hospital orderlies and attendants.

In the comparatively quiescent periods most of the cases seen by me (some thirty in all) conformed to the accepted views on this point. Mention of the state of consciousness introduces a fine distinction between "attention" and "interest." Granting an intact sphere of a consciousness, the average case shows an unquestioned ability to observe things, but an utter failure to concentrate the mind. When these patients are "talked at hard" for a short time, it is possible to get their half-hearted attention, and yet it is perfectly clear that they take absolutely no interest in what you desire to force upon them. No matter how eagerly they may want to entertain and absorb a thought, spontaneous animation is wholly lacking. In a dozen instances, patients were repeatedly up to a point where I thought their attention and inquisitive demeanor suggested interest, only to find sudden release from the momentary tension and entire disinterestedness.

The memory, speech anomalies, thought sequence, consciousness, and orientation may all be greatly modified by profoundly anxious and stuporous states as well as by the hallucinations and delusions that are so commonly present in this psychosis. The hallucinations are frequent in cases of acute or subacute onset, most often recurrent, but occasionally persistent. The auditory variety occurs more often than any other, visual and tactile following in their order. Kraepelin states these hallucinations tend to amuse rather than annoy the patients.

Delusions which are either fleeting or continuous have a greater significance. Coming on in the early stage of the disease, they partake of the negative emotional tone. Sadness is mingled with hypochondriasis, and ideas of sinning and being sinned against supervene. A disordered yet in a measure systematized type of delusion like and unlike that of true paranoia appears only in the dementia paranoides form of dementia præcox. The grandiose ideas and fantastic pictures prevailing for a time in the initial period of the disease are strongly reminiscent of the beginnings of paresis. The delusions of dementia præcox differ in the fact that they are readily influenced by suggestion, quickly remoulded—rapidly overthrown—not at all uniform, and lose their force entirely with the advancing mental deterioration.

Unsoundness of mind is in almost every instance foreshadowed by a change of disposition. The alteration of what Kraepelin denotes as the "Gemüth" and which for lack of a more satisfying term may be called "temperament," arouses even the lay mind to the first grave suspicion of impending mental disorder. A peculiar indifference, a growing disinterestedness which deepens into profound apathy takes firm hold of these sufferers. They cultivate solitude and remain unmoved by the concern of relatives and friends, who entreat them to take a more active interest in the affairs of every day life.

I have in mind a healthy, robust-looking, young, unmarried woman, Miss D., whose dementia was most unobtrusively ushered in with this preference for retirement. It first showed itself in a disinclination to remain at the dinner table with the family longer than was absolutely necessary, and the mother was the first one to take note of this. The patient had always participated in the leisure affairs of the family, did her share of home-entertaining, played the piano and sang for friends, was regarded as an adept at planning church entertainments, entered into the spirit of club sociables with vim and animation. In talking with her of this "change of heart" (to use her own expression), she said she was unable to ascribe any cause for it. Incidentally she spoke of having a pain in her side, which she said was at that moment rather severe and constant, but mention of it was not attended with the sort of expression that would indicate suffering or even physical discomfort. Asked about her growing distaste for sociability, she merely answered that she was glad of every opportunity to steal away to her room, and sit alone for hours at a time, because it was restful. Her interest in current books was on the wane; her ambition to excel in fancy and needle work was lost; her pleasure at cards was a thing of the past; her unwillingness to make new acquaintances was born rather of indifference than of aversion. Sitting with hands folded in her lap, staring vacantly into space for hours at a time, she could give no idea of the thoughts that possessed her. She was not dejected; she did not brood; she just did not know why she sat there so unconcerned about everything. All efforts on the part of her family to supply diverting entertainment were becoming more and more ineffectual. Her conversation was without play of feature, without facial token of emotionalism.

Frequently accompanying this morbid degree of apathy in dementia præcox, there are sudden outbursts of motor excitement, which fact the history of a young lad, aged seventeen, recently seen in the Neurologic Dispensary service at Northwestern University Medical School, will serve well to illustrate: The boy had incurred his mother's censure for his "new bad habits" of coming home from work at odd hours of the day, moping around the house, getting off into a corner or looking steadfastly out of the window, and occasionally muttering to himself. One day without warning, the boy got his discharge from the firm that had employed him for some two years, and upon inquiry into the company's action, it was stated that in one month he had been in three fights, precipitated so far as could be determined only by a sudden disposition on his part to hit somebody. He had never been guilty of this sort of misdemeanor at home, although his brooding spells frequently culminated in an unprovoked, senseless tirade against his mother.

The chief characteristics of such acts betokening motor excitement are their impulsiveness, speed, senselessness and varying degrees of violence. These inordinate phases of sudden temper, which lead to deeds of destructiveness and personal violence, remind one forcibly of the similar automatic, annihilistic acts of the psychic epileptic.

The clinical picture is incomplete without mention of that symptom termed "negativism," which stands out in such bold relief as a senseless stubborn resistance brought to bear against every measure of outward influence. This tendency is expressed both in the simplest mechanical movements and the more complicated acts of volition.

One patient who held his forearm half extended at the elbow would not allow me to flex it even the least bit, but on trying to place it in full extension, there was a quick surrender of the member to complete flexion. In another case the head and eyes had for several hours been deflected to the left side; with considerable force I was able to correct this position, and keep the head slightly turned to the right, but efforts to deviate it to the far right position immediately threw it into the original left pose, where it remained. Asking these patients to shake hands meets with a refusal, but not of the kind induced by unfriendliness or ill-will. Repeated requests may bring only stolid indifference, a laugh, or a "I won't," in answer—but not the hand. When they are up for the day, they strip off their clothing and run around naked—when they are put to bed at night they want their clothing on. If on refusal of food they require to be fed the next minute, they resist the feeding and eat of their own accord. When privileged to enjoy corridor life in institutions with other patients, they prefer solitude, and no sooner in isolation than they insist on companionship and sociability. Order these patients to bed and they remain up;—order them up and they remain in bed. These instances of opposing impulses, of negativism, occur alike in periods of excitement and stupor, always vary in degree and manner, are entirely disaffected by external influences and only capable of resolution, without apparent cause through the dictates of the inner will. Acknowledging the great difficulties encountered in the care of cases given to much of this negativism, it is gratifying to know that only the severer forms of katatonia display it in high degree and then not for long; but says Schüle, it is often noted as a prominent and lasting symptom in many of the climacteric melancholias.

It is not at all uncommon for the alienated to be so thoroughly dominated by the "force and frequency of impulses" as subconsciously to betray their content in manifold stereotyped movements (most distinctive in katatonia), mannerisms, mutism, verbigeration, echolalia, echopraxia and the Ganser symptom of "Vorbeideren" (circumlocution).

In the late stages of dementia præcox when the patients have run almost the entire gamut of symptoms and weakmindedness alone supervenes, then there is shown a disposition to do the things asked of them without offer of hesitation or resistance on their part. They come to be meek, mildmannered, tractable in the extreme and do things automatically; and some of the most unique features in this psychosis are referable to such automatism.

That the capacity for work suffers, that adaptation to new conditions grows more and more impossible, that constant urging is of no avail in initiating new tasks, and that only monotonous, rigidly routine work is faultlessly done, is the natural sequence of the progressive dementia.

The anomalies mentioned all or in part, may appear well accentuated in some of the writings of these patients, as has been shown by Clemens Neisser,[18] Kraepelin and others. Neisser has called attention to the special characteristics in the writings of the katatonias, and Kraepelin likewise dwells on some of the extravagant forms that embrace endless repetition of words, phrases and whole sentences. He comments on the wholly irrelevant, inane insertions that creep in between moderately sound thoughts, and dwells on the habit of mirror-writing cultivated in katatonia.

Not long ago I was privileged to see one of Dr. Hugh T. Patrick's patients, a girl of twenty, whose parents were so concerned about her rather sudden complete change of disposition and mentality, as to want her observed in a sanitorium. Some weeks of careful inquiry and observation under such auspices, warranted a diagnosis being made of incipient simple dementia. In due time the girl was taken home by her father; directly thereafter Dr. Patrick received for perusal the following letter addressed by the patient to a girl friend, and which I am permitted to insert here:—

"Dear Miss——

"Papa and I reached —— this morning about ten o'clock. I believe my younger brother, a lady from home and her young son were down to the train this morning to meet us.

"My sister and I took a walk this morning after I reached here. She gave me some nice presents, two handkerchiefs which she made, and the presents which had been sent here from home. There was a present here from one of the girls I was in school with at home, and from three of the girls I was away at school with I received nice presents, and a nice present was sent to my sister and I, by one of the girls who is away at school now.

"My younger sister gave me a nice present which she had made. Papa brought two presents down in his trunk for me, one nice present was from the wife of my cousin who came to C. and the other nice present my cousin who came to C sent.

"My younger brother gave me a present. I will close now. Write to me when you have time.

" Yours"

(Signature)

It is difficult to think of this letter as coming from a seminary student twenty years old. The repeated allusion to receipt of presents in simple, short, unembellished sentences; the word "present" appearing ten times, and the phrase "nice present" recurring six; the puerile treatment of every thought and the dearth of ideas certainly indicate a state of considerable mental bankruptcy.

Although entirely subordinated to the psychic symptoms and their relationship to the underlying disease not altogether well understood, the somatic manifestations of dementia præcox are none the less notable and deserving of brief mention. In consonance with the statements of all authors concerning the tendon reflexes, I found them greatly and uniformly increased in all the cases observed. There was no suggestion of that variability of the reflexes shown in paretic dementia, in which they appear as normal, diminished, increased or lost. The patellar responses were nearly always brisk, amounting to a quick somewhat spastic extension of the whole leg, and inequalities were not noted. The Achilles-jerk was elicited as normal or increased, never decreased.

Interest also attaches to the pupils, which in every instance were uniformly and widely dilated. Piltz[32] in the *Neurologisches Centralblatt,* August 1, 1903, (Der diagnostiche Werth der Unregelmässigkeiten des Pupillarrandes bei den sogenannten organischen Nervenkrankheiten) mentions the pupil of a dementia præcox in his consideration of the iris irregularities in organic nervous disease, and states that aside from the dilated pupil he has noted in several katatonias an irregularity in the periphery of the iris, which shifts from one quadrant to another. He further states that "pupillary unrest" (Die pupillen Unruhe, von Lacquer) so clearly and convincingly demonstrable under normal conditions, indicating an easy susceptibility to sensory stimuli first passing through the central nervous system, and disappearing in such disease as tabes and general paralysis, is absent in præcox. A wholly incontestable explanation of this absence of unrest is not as yet at hand except that we are satisfied to regard it as an expression in part of the great quantitative reduction in psychic acumen.

It has also been determined that in dementia præcox the sensitiveness to cocaine is reduced, but is normally retained to homatropine and pilocarpine. The so-called orbicularis reaction of the pupil described first by Albrecht von Graefe and recently revived by Westphal and Piltz is much more difficult to elicit in this disease than in normal individuals.

The epileptiform convulsions and apoplectiform fits mentioned by Kahlbaum in his first contributions to this subject have been accorded a place in the physical symptomatology by all subsequent authors, but in the few patients at my disposal, I was able to note the history of a seizure in only one, a hebephrenic. That convulsive movements, facial twitchings, and paralyses, occur commonly in the katatonic variety is conceded. Kraepelin states that convulsive attacks large or small occur in about 18 per cent. of all cases, and attack women nearly twice as frequently as men, but owing to a small material I was unable to convince myself on this point. In one young girl, I observed peculiar irregular, widely excursive movement, localized in the right hand, that bore closest resemblance to the "athetoid" variety; and from Kraepelin we learn that these "choreiform movements" in some of the patients may persist for a long time. Hysteroid convulsions, aphonia, hiccough and aphasia were not met with, but vasomotor disturbances, cyanosis, profuse hyperidrosis, excessive drooling and dermatographia occurred in a number of cases, more especially in the katatonias. The bodily temperature was normal or subnormal. Inquiry into the sleep of patients capable of appreciating this factor elicited the fact that it was frequently disturbed and fraught with dreams. The appetite was subjected to fluctuations from one extreme to another, instanced by either the refusal of food, moderate indulgence, or voraciousness and gluttony. Although obesity is not an uncommon development in the course of the disease, the general tendency is quite the other way and the patients lose weight to a degree of emaciation.

In attempting a sharp differentiation of clinical forms, I recall Kraepelin's expression of embarrassment in venturing a classification based on strictly scientific premises, wherein he admits there is a whole series of intermediate forms, which differ so markedly from each other and from the greater types that assignment to any one group is undesirable and untenable; he adds that his arrangement is cardinally for the purpose of simplifying the little knowledge already acquired. In the concluding lines of his chapter on a general view of dementia præcox, Kraepelin divests himself of the hopeful thought that a more exact knowledge of the nature of the disease will reveal an entirely new point of view on which to build a finer and stronger clinical superstructure.

It is regrettable that a discussion of the hebephrenic, katatonic and paranoid types necessarily entails a tedious review of much that has already been set down in the general symptomatology and much that is reserved for the consideration of differential diagnosis. Meyer's point "that the forms are very variable and a division into groups is artificial and claims only didactic value" is well taken, but types are desirable since they serve so well to facilitate our understanding of distinctive symptoms appearing early in the course of the disease long before the polymorphous picture comes into full view or ultimate mental obscuration has taken place.

Hebephenia—Hecker is responsible for the creation of this term, by which he meant to convey the idea of a mental disorder occurring at the age of puberty—an insanity of pubescence. Since the inception of that word there have always been a few who were inclined to forget that every insanity developing at the age of puberty must not necessarily be a pubescent insanity in Hecker's sense of the term. Seeing the chance for increasing error on this point, Kraepelin offered the term Dementia Præcox in justification of his view that this disease attacks those well removed from years of puberty, individuals who have arrived at the third and even fourth decade. Thus the co-existent terms Hebephrenia and Dementia Præcox that appear in nearly all writings of the past ten years, except Kraepelin's, may be construed as interchangeable and synonymous.

Kraepelin lets "dementia præcox" appear as the disease name and "hebephrenia" as one of its varieties. Since excellent observers have taken the stand that hebephrenia and katatonia merge too readily to warrant separate mention and seek to discredit the existence of the former as a clinical type, we are constrained to refer again to Kraepelin, who, despite his acknowledgment of transitional forms and abortive types up-holds the division and treats it accordingly in his latest edition on clinical psychiatry. As stated by him at the outset of his treatise, he sees much to commend in Daraszkiewicz's enlargement of Hecker's original idea. Hecker was wont to consider hebephrenia as a condition ushered in by melancholia, followed by a period of mania and rapidly ending in a peculiar weak-mindedness. Since this narrow conception so quickly excluded untold numbers of similar cases, Daraszkiewicz ("Ueber Hebephrenie insbesondere deren schwere Form"; Dis. Dorpat, 1892), sought to include in the definition the "severer forms," by which he meant those leading to profound dementia. Kraepelin retains this version in the recent editions of his Psychiatry.

That heredity is a very frequent and important causative factor is an accepted dictum, but the figures vary in ratio to the unreliability of the testimony on this point. Kraepelin thinks that heredity is an influence in 70 per cent. of ascertained cases. Although hebephrenia is occasionally engrafted on cases of mild congenital idiocy, it very much more often builds on sound cerebral soil. The proportion of asymmetrical skulls and faces, the number of stigmatized individuals with malformed palates, abnormally set ears, left-handedness, stammering and the like is very small. That an infectious agent enters the economy by one or many routes and exerts a toxic influence on the highly organized tissues of the brain is altogether improbable. Dementia Præcox is a psychosis of endogenic origin.

The symptoms are multiform and in a large proportion of the cases begin abruptly, but there are the lighter forms, which despite their great forensic importance are overlooked or under-estimated. Hebephrenics are recruited from all strata of society and the magnitude of their acts alone proclaims them as either flagrant or mild offenders of the social code liable to total ostracism or bare tolerance. Young men of undoubted ability are satisfied to become copyists, some want nothing better than clerkships, others become low salaried care-takers of great interests that once they aspired to direct as their own.

Continental writers tell us that hebephrenia with its onset at about the time for application to the service is greatly in evidence in the European militaire, and that the symptoms whenever detectable make the applicants ineligible. A canvass of work-shops, jails, charitable institutions and service bureaus would bring to light a class of individuals, so-called creatures of circumstances and misfortune, tramps, vagabonds, indigents, fakirs and confidence men, who have allowed the better prospects of life to go by default, not owing to any inherent or instinctive passions for doing the things that are wrong and reprehensible, but because of the blighting touch of mental disease laid upon their shoulders in the young years of promise by the unseen hand of an unkind fate.

To emphasize this class, who are admittedly below par mentally, dull of comprehension, indolent, self-complacent, self-satisfied, ill-tempered and erratic yet harmless and beyond the pale of medical or legal adjudication, is not so much my purpose as to dwell upon a coterie of individuals, whose lives are burdensome in any environment, whose future is vested in institutional care until the end. These sufferers first complain of headache, vertigo, sleeplessness and a sense of depression, the oppressiveness of which is only relieved by sudden induction of an expansive period, during which there run riot an unbridled exhilaration, restlessness and egotism.

In rapid succession, brazenness is exchanged for modesty, loquacity for silence, gayety for moroseness, irritability for sullenness. More than this the relatives and friends notice sudden eccentricity of manner and new methods of address, confusion of speech, refusal of food, derision of friends and disavowal of obligations. From a condition of thoughtlessness, indecision and apathy, the patients pass suddenly or by stages into fits of maniacal violence, during which they give vent to terrible outbursts of anger and then sink back completely exhausted into depression and sadness, enthralled by active delusions and controlling ideas of self-abasement and great unworthiness.

Although this sequence of the positive emotional tone succeeding the negative, with perhaps a stuporous period intervening, is the rule, there is no cyclic character implied as for instance in circular insanity. The irregularity in this respect is somewhat responsible for the tendency to call these cases mania, confusional insanity, melancholia, according to the evident phase of the process.

What has been said in the foregoing paragraphs of orientation, judgment, memory, thought-sequence, interest and attention applies with equal force here. That with depression we frequently have mutism, verbigeration, negativism, uncontrollable, unprovoked laughter, peculiar movements, attitudes, stupor and catalepsy (flexibilitas cerea) is not controverted, but their presence robs hebephrenia of its purity of type. After the several cycles referred to have spent their force then the mental deterioration manifests itself prominently. Kraepelin says 75 per cent. attain to a high degree of dementia. With its development and progress comes the habit of sitting alone, drooling saliva, passing excreta in the clothes, feeding voraciously or not feeding at all, merely vegetating; it is the same horrible picture presenting in the terminal stages of general paresis. About 17 per cent. of the cases come to a halt long before this stage; one or two stormy periods over with, and the patients turn quietly to the performance of such routine duties as will gain them a livelihood. Of the cases which Kraepelin was able to follow for a long time, eight per cent. lost all traces of their symptoms and might be called recoveries, but he does not omit to add, that many individuals may be able to keep abreast of the work expected of them, maintain a relatively good social balance and nevertheless remain stigmatized as cases of hebephrenia.

CASE REPORT I—C. H., male, aged twenty years, nativity U. S. A. Born of a German father and American mother. He enjoyed a high school education and is by occupation a musician. At time of the patient's birth, his father was thirty-three and his mother twenty-nine years old. He is 5 ft. tall and weighs about 115 lbs.

Personal Characteristics—He was placed under institutional care May, 1904. The only assigned cause in his commitment papers was overwork, excessive musical study. From these papers, I ascertained that he was a boy of quiet, unassuming disposition, with no vicious habits, whose health had always been very good until about February, 1904, when he showed marked nervousness and fear. It was alleged by the family and friends that his behavior had for some time past been queer. Above all else he was stubborn and obstinate. Told to do one thing, he insisted upon doing the opposite. He proved resistant to all measures proposed by his friends. Although he had always been quiet and retiring in his demeanor, there is no evidence of his having experienced periods of regular depression. An attempt on an April night to strike his mother caused the family to think him insane. No other acts of violence or destruction are reported.

Physical Condition—The patient was short in stature and of fair complexion. The configuration of the head was notably asymmetrical, dolicocephalic, elongated in the antero-posterior diameter, with a wide square brow, long face, full, well-rounded jaw with long prominent chin. (See photograph Fig. 1.) The face was expressionless and the eyes given to

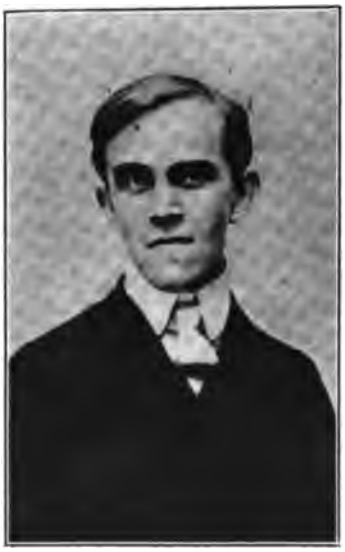

Fig. I, Case 1—C. H., aged 20,
Hebephrenic type

constant vacant staring at far off space. He had a large facial mole. The open mouth revealed an extremely high arched narrow palate. There were no objective findings on the part of the muscular system, no history of convulsive movements or twitchings. The tendon reflexes especially at the knee were exaggerated, and the pupillary reflex was very active in the presence of widely dilated pupils. The viscera were negative. The appetite was poor, the tongue coated and the bowels constipated. There had been for some time a tendency toward incontinence and he had soiled his linen with feces before entering the hospital. No disturbance of special senses were noted.

The mental phenomena speak rather frankly, I think, for the distinctive type of the psychosis under consideration. Subjecting the patient to cross examination it was difficult to get his attention and almost impossible to get his interest. The slightest move served to distract him. His answers came without play of facial feature, in almost monosyllabic utterance and were dull and stupid. The tonal quality of the voice was weak and monotonous.

The patient exhibited the peculiar type of memory-defect so characteristic of this form of dementia in that he recalled with exactness the opus of almost every musical work he had played and studied for years back (a most remarkable feat of memory even for the normal mind) and yet could not say how long he had been in the hospital, nor remember any of the occurrences of his hitherto brief residence there. He said he was born in 1884, but did not know the present day or date of the month. He spoke both German and English, but his conversation was bland, colorless in both.

At the time I saw him, he gave no evidence of delusions, but his history stated that he thought he was "Jesus Christ and that his father was Almighty and possessed of eternal age." At one time after some weeks in the hospital, he became restive, then excitable beyond control, denunciatory in speech and destructive in action; these symptoms culminated in one maniacal outburst, after which he lapsed back into a state of grave depression.

(*To be continued*)

THE EARLY OCULAR SIGNS OF DEMENTIA PARALYTICA.

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There is no dearth of statistics in regard to the ocular signs of paresis. Many of the reports, however, are open to criticism in two respects: first, because patients in different stages of paresis have been included, so that late as well as early conditions are put in the same category; and second, because frequently no effort has been made to exclude cases complicated with tabes, so that the pupillary, muscular and optic-nerve symptoms of tabes are added to the eye symptoms properly belonging to paresis.

It has, therefore, seemed to me worth while to keep notes of the eye conditions found in a series of cases of paresis uncomplicated with tabes (*i.e.*, having exaggerated knee-jerks) examined at the Manhattan State Hospital within a short time after their admission. These patients were thus seen fairly early in the disease, yet all had been under observation long enough to rule out alcoholic pseudoparesis and to permit an absolute diagnosis of paresis to be made.

In the subjoined table are recorded the ocular conditions of seventy paretics in an early stage of the disease, showing how far pupillary disturbances may be of value in diagnosis.

The method of examination employed is the following: The patient is seated eight feet from a single window with a dark shade drawn over the upper sash. A north window furnishes the most constant light, and there should be no other source of illumination in the room. The patient should face this window squarely, otherwise the more strongly illuminated pupil may contract more than the other, because the consensual reaction is not always equal to the direct. After he has looked out of the window for a minute or two the patient is directed to look up at the ceiling. Since less light then enters his eyes, his pupils dilate. Even with sluggish pupils the light reaction is well made out when the eyes are directed alternately from window to ceiling after remaining a minute in each position. But if the pupils are very sluggish, perhaps the best means of eliciting the light reaction is to have the patient first face the window and then turn his back to it. This is done with both eyes open and then with each eye closed.

The advantage of examining the pupils with the gaze directed toward the ceiling is that the reflex from the cornea does not lie over the pupil and obscure it. Any irregularities in shape can then be made out, and it is easy to measure the size of the pupils. While the patient is looking up the consensual and direct reactions of each pupil may be obtained by covering and exposing each eye singly, with the other eye first open and then covered. With sluggish direct action we may expect sluggish consensual reaction, but rarely a loss of consensual reaction, as is sometimes stated.

In the subjoined table *each pupil has been measured* with the pupillometer *while the other eye was covered*, thus excluding any consensual contraction. Hence, many of the figures are larger than if the pupils had been measured, as they often are, with both eyes exposed.

In order to elicit the sensory reaction the skin of the neck is pinched several times in rapid succession, or the thumb nail is drawn sharply across the temple several times, when each pupil should dilate slightly. There are some sources of error in making this test. It has been advised to have the patient fix the observer's eye while the test is being made. The reaction so obtained is often well-marked, but one cannot be sure that the dilatation is not due to a relaxing of the accommodation due to distraction of the attention when the skin is irritated. Personally, I prefer to make the test with the patient looking at a point on the ceiling. After the gaze has been directed upward a few seconds a spontaneous dilatation of the pupil often occurs, or there may be a continued oscillation or hippus, and this is particularly frequent in paretics. Hence the pupils should be watched until they are entirely at rest before the test is attempted, or better, the irritation of the skin may be continued for 20 or 30 seconds, and when the irritation is stopped the pupils will be seen to contract if sensory dilatation has occurred during the irritation.

Physiological variations of the pupils must be taken into account. Other things being equal, the pupils are somewhat larger in blue eyes than in brown. They are often quite large when the patient is myopic and small when he is hyperopic, because the myope accommodates little and the hyperope accommodates excessively. Age and hardness of the vessels also have their influence. In childhood the pupils are large, in early adult life medium-sized, and in middle age smaller. Thus, in a series of normal eyes in persons between 25 and 35 years of age the pupils measured with one eye closed ranged from 4.5 to 6 mm., the average being 5.25 mm., and in a series between the ages of 40 and 50 the range was from 3 to 4.5 mm., the average being 3.75 mm.

The sensory reflex is usually well-marked in early life, but in middle age it is slight, and in older persons it is often absolutely wanting.

The shape also varies with age. In early life the pupil is round, but in middle age some irregularity of the pupils can usually be made out if the ophthalmoscope is used or the irregularity may be sufficiently pronounced to be seen with the naked eye.

Inequality of the pupils is too common to have much pathological significance. An excitation of the cervical sympathetic will cause a dilatation of the pupil on the same side, which may last a few minutes, or days, or even years. Similarly, a contraction of one pupil may be brought about by a spastic contraction of the sphincter pupillæ. Considerable attention has been paid recently to alternating or see-saw mydriasis (Springende Pupillen, Mydriase à bascule), which is seen in paresis as well as in other affections of the nervous system. In these cases the relative size of the pupils may change even in the course of the examination, and with the alterations in the size of the pupils there may be alterations in the sensory and light reactions. Hence, repeated examinations are desirable, for in paresis particularly the condition of the pupils sometimes differs noticeably from day to day.

In the subjoined table the refraction, as determined by retinoscopy, is recorded in dioptries, E. indicating emmetropia, H. hyperopia and M. myopia. The refractive error was sufficient to account for any diminution that existed in acuteness of vision, except in two cases in which there was disseminated choroiditis. In one other case there was slight blurring and pallor of the disc without diminution of vision, and in another case there was a posterior synechia.

The interior of the eyes was normal except in these four cases, in which doubtless we had to do with the residua of syphilitic inflammations.

It is frequently stated that optic-nerve atrophy is present in a considerable percentage of cases of paresis, at least in the late stages of the disease. Among these 70 patients there was no case of optic-nerve atrophy, and among some hundreds of patients examined at various stages I have rarely found pallor of the optic discs except when symptoms of tabes or of nicotine poisoning existed.

SUMMARY.—The *shape* of the pupils was perceptibly *irregular* in 51 patients, or 70 per cent., an abnormally high percentage considering the ages of the patients.

Inequality of the pupils was found in 32 patients, or 45 per cent.

The *sensory* pupillary reflex was absent in 61 patients, or 87 per cent., a very high percentage considering the ages of the patients.

The *direct light reaction* was perceptibly *sluggish* in one or usually both eyes in 15 patients, or 21 per cent.

The *direct light reaction* was entirely *wanting* in one or usually both eyes in 20 patients, or 28 per cent.

The *convergence reaction* was sluggish in 6 patients, or 9 per cent., in all of whom the light reaction was sluggish or wanting.

The *size* of the pupils ranged from 1.5 to 5mm., and in 37 patients, or 55 per cent., one or both pupils were *smaller* than the average size for the patient's age and refraction.

As a rule, with Argyll-Robertson pupils the sensory reflex is wanting, and there is spastic myosis, the pupils being small; but as the paresis or tabes causing the Argyll-Robertson pupils progresses the spasm of the sphincter pupillæ relaxes and the pupil becomes large, and the convergence reaction becomes sluggish or disappears. In this series among the 20 cases in which the light reaction was lost, in 7 cases one or both pupils were

4 mm. or more in diameter, but it is likely that they had been small earlier in the disease.

In a certain number of cases of paresis the pupils remain normal in every respect up to a late stage of the disease. And it should be said also that in alcoholic pseudoparesis we may find all the pupillary changes found in true paresis, so that the examination of the eyes may not help one in distinguishing between true and pseudoparesis.

RECAPITULATION —As this table shows, in true, uncomplicated paresis there is early in the disease almost constant absence of the sensory reflex, in half the cases irregularity of the pupils, in nearly half inequality of the pupils, in more than half abnormally small pupils, in a fifth of the cases loss of light reaction, in another fifth marked sluggishness of light reaction, and in a few of those with diminished light reaction a diminution of convergence reaction also.

While, as stated above, absence of the sensory reflex, myosis and irregularity of the pupils may not be of much significance in persons past middle life, they are of considerable diagnostic importance when they occur in earlier life; and Argyll-Robertson pupils are, of course, of great significance whatever the patient's age may be.

In conclusion I wish to express my thanks to Dr. E. C. Dent, Superintendent of the Manhattan State Hospital, and to many other medical officers of the institution for affording me every facility and assistance in making these examinations.

					Pupils.					
						Size, mm.		Reflexes.		
Sex.	*Age.*	*Vision.*	*Refraction.*	*Fundi.*	*Shape.*	*R.*	*L.*	*Sensory.*	*Light.*	*Convergence.*
F.	37	20-20 each	E.	Normal	Irreg.	4.5	4.	Present	Good	Good
F.	36	20-20 each	E.	normal	irreg.	3.	3.	absent	sluggish	good
F.	41	20-20 each	R. H2, L. H3D	normal	round	4.	4.5	absent	good	good
F.	36	R. 20-40, L. 20-50	M. Ast.	normal	irreg.	3.5	3.5	absent	sluggish	good
F.	39	R. 20-30, L. 20-20	H2D	normal	irreg.	2.5	2.	absent	absent	good
F.	54	20-20 each	E.	normal	irreg.	1.5	1.5	absent	absent	good
F.	25	20-20 each	H1.5D	normal	round	5.	5.	absent	sluggish	good
F.	52	20-30 each	E.	normal	round	2.5	3.	absent	absent	good
F.	35	20-20 each	E.	normal	irreg.	3.	3.5	absent	R. good, L. sluggish	good
F.	38	R. 20-200, L. 20-70	H7D	normal	irreg.	3.	2.75	absent	sluggish	good
F.	32	20-20 each	H1.5D	normal	irreg.	3.	3.5	absent	good	good
F.	49	R. 3-200, L. 20-200	H.	choroiditis	round	3.	3.	present	good	good
F.	24	20-20 each	H1.5D	normal	irreg.	4.5	4.5	absent	sluggish	good
F.	45	20-20 each	H1D	normal	irreg.	2.75	3.	absent	sluggish	good
F.	47	R. 20-70, L. 20-200	R. H1D, L. H & Ast.	normal	irreg	2.75	3.	absent	sluggish	sluggish
F.	42	20-70 each	H2D	normal	irreg.	2.5	2.5	absent	absent	good
F.	32	20-30 each	H.Ast.	normal	irreg.	5.	5.	absent	good	good
F.	29	20-20 each	H1.5D	normal	irreg.	3.	3.	absent	absent	good
F.	41	20-20 each	E.	normal	round	4.5	4.5	absent	good	good
F.	37	20-20 each	H1.5D	normal	irreg	3.5	3.	absent	good	good
F.	36	20-20 each	E.	normal	irreg	3.	3.5	present	good	good
F.	46	R. 20-50, L. 20-70	H.Ast.	normal	round	2.5	2.5	absent	absent	good
F.	36		Mix.Ast.	chloroiditis	round	4.5	4.	absent	absent	good
F.	45	20-40 each	H2D	normal	irreg.	4.	3.5	absent	good	good
F.	28	20-20 each	E.	normal	irreg.	4.	4.	absent	good	good
F.	37	20-30 each	H.Ast.	normal	irreg.	2.5	2.5	absent	absent	good
F.	46	20-20 each	H1D	normal	irreg.	3.	3.5	absent	good	good
F.	25	20-30 each	H1.5D	normal	round	3.5	3.5	absent	good	good

F.	42	20-50 each	H2D	normal	irreg.	3.5	3.5	absent	good	good
F.	37	R. 20-40, L. 20-50	R.H2D, L.H2 & Ast.	normal	irreg.	4.	4.5	absent	good	good
F.	40	20-70 each	H2.5D	normal	round	4.	4.	present	good	good
F.	43	20-20 each	H1D	normal	round	5.5	5.5	present	good	good
F.	34	20-20 each	H2D	normal	irreg.	posterior synechia	4.5	absent	good	good
F.	42	20-20 each	H1D	discs pale & blurred	irreg.	3.5	3.5	absent	good	good
F.	40	20-20 each	H2D	normal	irreg.	4.	4.5	absent	sluggish	good
F.	37	20-20 each	E.	normal	round	4.	4.	present	good	good
F.	40	20-20 each	E.	normal	round	4.	4.	?	good	good
F.	35	20-20 each	H1D	normal	irreg.	1.5	2.5	absent	absent	good
M.	55	R. 20-50, L. 20-50	R. H1D, L. H. Ast.	normal	irreg.	5.	3.	absent	sluggish	sluggish
M.	29	20-20 each	E.	normal	round	5.	5.	absent	good	good
M.	45	20-70 each	M2.5D	normal	irreg.	2.	2.5	absent	absent	good
M.	43	20-30 each	H2D	normal	irreg.	3.5	3.5	absent	good	good
M.	40	20-20 each	E.	normal	irreg.	3.5	4.	absent	absent	good
M.	32	20-20 each	E.	normal	round	4.	4.	present	good	good
M.	24	20-20 each	H1D	normal	irreg.	3.	3.	absent	good	good
M.	42	20-30 each	E.	normal	irreg.	4.	4.	absent	good	good
M.	42	20-20 each	H1D	normal	irreg.	3.5	3.5	present	good	good
M.	33	R. 20-30, L. 20-20	E.	normal	irreg.	4.5	3.5	absent	good	good
M.	24	20-20 each	E.	normal	irreg.	4.	4.	absent	absent	good
M.	58	R. 20-50, L. 20-70	H1.5D	normal	irreg.	1.5	2.	absent	absent	good
M.	26	20-20 each	H1D	normal	irreg.	4.5	4.	absent	absent	good
M.	41	20-20 each	H1D	normal	irreg.	4.5	3.	absent	absent	good
M.	27	R. 20-70, L. 20-20	R. H.Ast.	normal	round	3.5	3.5	absent	sluggish	good
M.	29	20-20 each	E.	normal	round	5.	5.	absent	good	good
M.	45	20-20 each	E.	normal	irreg.	4.	3.5	absent	good	good
M.	55	R. 20-100, L. 20-20	R. Ast., L. H1D	normal	irreg.	3.5	3.5	absent	slugggish	sluggish
M.	42	R. 20-20, L. 20-40	E.	normal	irreg.	4.	4.	absent	sluggish	good
M.	35	20-20 each	E.	normal	irreg	3.5	3.5	absent	good	good
M.	27	20-20 each	E.	normal	irreg	3.5	3.5	present	good	good
M.	55	20-70 each	H1D	normal	irreg.	5.	3.	absent	absent	good
M.	39	20-30 each	M.Ast.	normal	irreg	4.	2.5	absent	absent	sluggish

					Pupils.					
						Size, mm.		*Reflexes.*		
Sex.	*Age.*	*Vision.*	*Refraction.*	*Fundi.*	*Shape.*	*R.*	*L.*	*Sensory.*	*Light.*	*Convergence.*
M	50	20-20 each	R. E., L. H1.5	normal	irreg.	4.	3.5	absent	sluggish	sluggish
M.	32	20-20 each	E.	normal	round	4.5	4.5	absent	good	good
M.	44	R. 20-40, L. 20-20	R. M1D, L. E.	normal	irreg.	3.5	3.	absent	good	good
M.	43	R. 20-40, L. 20-20	R. H1D, L. E.	normal	irreg	2.5	3.	absent	R.present, L. absent	good
M.	37	20-100 each	H2D	normal	irreg.	3.	3.	absent	good	good
M.	55	20-20 each	E.	normal	round	4.	4.	absent	good	good
M.	45	20-20 each	E.	normal	round	3.	3.5	absent	R. sluggish, L. good	good
M.	42	R. 20-20, L. 20-70	R. E., L. M1D	normal	irreg	3.	3.5	absent	good	good
M.	35	20-20 each	E.	normal	irreg	2.	2.	absent	absent	sluggish

PSYCHASTHENIA: ITS CLINICAL ENTITY ILLUSTRATED BY A CASE.*

(Preliminary Paper.)

By Sidney I. Schwab, M.D.,
of St. Louis, Mo.

In this paper an attempt will be made to give a more exact definition to the term Psychasthenia, and to call attention to the work of Pierre Janet in this connection and to illustrate some of the ideas here set forth by a brief description of a case which typifies, in a somewhat exaggerated form, perhaps, what is meant in a clinical sense by the word Psychasthenia. Its justification to a clinical entity in the classification of the so-called neuroses is a matter which usage supported by the consensus of the best neurological opinion alone, can decide.

It will not be out of place to mention at this point that Professor Janet's visit to this country last year, at the time of the Congress of Arts and Sciences at the Exposition at St. Louis gave an opportunity to hear at first hand his views upon a subject in neurology which he has made particularly his own. I count it no little honor to be able to bring before you the effect of some of his work, and to be able to illustrate by the recital of a case which he himself, in conjunction with Dr. Putnam of Boston, Dr. Fry and myself saw. This case Janet unhesitatingly pronounced to be psychasthenia instead of hysteria or neurasthenia, or a combination of both.

At the clinical meeting of this society last year in St. Louis I had the pleasure of presenting this case, and the contention was then made by some of those present that there was little justification for considering it other than one or both of the abovementioned diseases.

It is apparent that multiplication of terms is entirely useless unless by it some advance in knowledge is obtained, and yet no evidence of the tendency toward such an advance is more striking than the desire to limit more exactly the meanings which are implied in the terminology of diseases.

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*Read at the meeting of the American Neurological Association, June 1, 2 and 3, 1905.

A valuable contribution to this end has been made by Dana in regard to neurasthenia, and a like attempt in respect to hysteria would result in a like increase in our power to counteract its effects.

The fact that we have at present no adequate conception of the term hysteria makes the task all the more difficult, and the fact that we are not likely to find one points unmistakably to the conclusion that too many types of diseases and too many variations of clinical pictures of diseases are without much thought classed as hysteria. Perhaps if it were possible to exclude certain conditions which are not so definitely hysterical but are psychogenic in their origin, it would be a step in the right direction of finally determining just what we mean by hysteria; or if this is not possible, of so limiting the term that it will lose much of the vagueness with which tradition has involved it.

There are a certain number of cases met with now and then for the explanation of which a diagnosis of hysteria leaves us dissatisfied. In such cases there are to be found no anomalies of sensation and none of the so-called stigmata of hysteria. No objective symptoms commonly associated with hysteria are present, and yet in extreme cases such patients may become permanently incapacitated from taking part in the activities of their former manner of living. In extreme examples of this condition, in which is included the case which forms the subject of this paper, a paraplegia develops in conjunction with a paralysis or paresis of most of the voluntarily controlled muscles. Underlying such a case will be found a mental state that is seldom found in hysteria. The fundamental characteristic of this state of mind from the point of view of differential diagnosis is its perfect preparedness for the symptoms as they appear, and a very accurate perception of why they do occur.

Whatever conception of hysteria we may choose to incline to and however broad we may make the meaning of the term neurasthenia, such a case leaves us without a satisfactory diagnosis, unless we assume the existence of another category of functional diseases. If this is granted, then the intimate psychology of the development of the morbid state of mind which underlies the condition may be capable of a somewhat clearer analysis.

In the introduction to his notable book "Obsessions et Psychasthenie" Janet sets forth his ideas in respect to psychasthenia in part as follows:

"The diseases which form the object of this new study are the obsessions, the manias, the folie du doute, tics agitations, phobies, delires du contact, states of anxiousness, neurasthenias, the bizarre sensations of strangeness and of depersonalization often described under the term cerebrocardiac neuropathy, or Krishaber's disease. They have sometimes been classed under the term degenerate deleriants, or neurasthenics, or phrenasthenics. I have often designated them under the term doubters "les scrupuleux," because doubt constitutes an essential characteristic of their thought, and under the more precise term psychasthenic, which appears to me to express feebly enough, it is true, the weakness of their psychical functions. This collection of diverse symptoms will permit the unification of many different diseases into one, and it may be possible to construct a great psychoneurosis modeled after that of epilepsy and hysteria. This will be psychasthenia, and will take the place of the obsessions, manias, phobies, insanity of doubt, of contact and cerebrocardiac neuroses."

This might seem to be an ambitious attempt, and indeed from a clinical point of view it is; but nevertheless, for Janet's purpose, which in this work at any rate is to submit these various states to the most minute psychological analysis, it proves distinctly feasible.

For the purpose of the present paper such a classification is certainly too extensive, and the one case which is to be its illustration would be but a slender prop to support so large a clinical differentiation. The much more modest purpose of submitting this case and leading you to see that it justifies the term psychasthenia will be all that will be attempted.

If this much is admitted, then there is hope that the term will be more exactly used with a more definite distinction from the diseases it might be confused with.

This case has been the subject of study and almost constant observation for a period of six years. The patient is a physician, and for that reason has himself been interested in the case as a medical problem. Consequently it has been possible for me to see more of the case than is usual with patients of this kind.

In brief, the outline of the case is as follows:

Dr. G———, forty-three years old, comes of a very neurotic stock. His mother has been a nervous invalid for many years, with attacks of mental depression, alternating with excitement and delusions more or less fixed. By the family physician this condition has been called hysteria. A sister and brother have been under my care for functional troubles of various kinds. In fact, there is scarcely any member of the immediate family that has not shown at one time or another some evidence of the neurotic strain mentioned above. The mother died a few months ago, apparently as a result of an apoplectic stroke.

The patient had always been considered a somewhat delicate child. In his school work he was always successful, and was looked upon as an unusually gifted boy in point of intelligence. As he grew to maturity there was very early to be observed two opposing forces in his mental make-up, in respect to his rapidly-growing ethical consciousness, a profound sense of duty and a distinct disinclination, amounting in some instances to physical repugnance to carrying out the conclusions which this sense made so obvious to him. Added to this was a strong religious inclination which seemed even this early to form a background to much of his deeper thought.

Of previous sickness other than the one antedating the beginning of his present trouble there need nothing be said except that there was always a lack of physical, though never of mental, vigor.

Coming to this country at the age of 25, he found that the knowledge of law gained in his university was of little service to him in the legal profession. He determined then to study medicine, as it appeared to him that in medicine the surest and most rapid advance might be made. It is to be observed that he had a natural repugnance to the study of medicine, but his sense of duty compelled him to this decision. He was a good student, graduating high in his class. He obtained the usual hospital position, and after his service was completed he decided to spend a year as assistant in the St. Louis Insane Asylum.

Again in this instance he followed the dictates of what he conceived to be his duty against his most evident disinclination, for he had a morbid fear of the insane and a most intense distaste for mental diseases His work there was very satisfactory to all concerned but himself, and he left very nearly exhausted by the struggle.

In practice he obtained a fair measure of success, fighting always against the mental state mentioned before. He represented in his practical work a man doing work well for which he had neither taste nor the consciousness that it was the work he could do best. After several years of general practice he decided to specialize in nose and throat. His reason for doing this was a hope that in the limitation of his activity he might obtain the peace of mind which had so long been denied him. He was active and successful in his new field until an attack of influenza in December, 1895, caused his final breakdown. Against this he struggled at the advice of his physician and attempted various sorts of exercise in the hope of overcoming a paralysis of his will, which his physician explained to him was the cause of his gradually increasing difficulty in moving about. This explanation plays an important part in the subsequent history of the case forming the nucleus of an idea which became more and more fixed as time went on.

Very gradually, but progressively, Dr. G—— lost the power of voluntary control of the muscles of the four extremities until he became a bedridden invalid or a prisoner in an invalid chair, unable to help himself, at times unable to move a single voluntarily-controlled muscle in his body. After a year spent in a hospital in St. Louis and another year at a sanitarium in Michigan he returned to St. Louis to the Baptist Sanitarium, where he has been ever since, practically in the same condition. In the summer of 1899 I saw him for the first time.

A physical examination made at that time and repeated many subsequent times showed an absolutely intact nervous system, except the total paraplegia of all four extremities. All reflexes were normal. There was perfect control of the sphincters. There was an intense atrophy of all the muscles, but so evenly distributed that disuse could satisfactorily explain it. The electrical examination showed normal conditions. There was no objective sensory symptom of an abnormal kind to be found. There was marked emaciation, but no more than would be natural in a case of this kind. The urine, blood, heart, lungs, etc., were normal. From the subjective side there was a collection of the most bizarre and curious paresthesia. It is sufficient to say that recently a physical examination showed practically the same results except that there had developed a marked emphysema of both lungs, totally obscuring the heart's area. It is probable that this is due to the superficial breathing which the patient has had for so many years.

During the time of my observation of the case the patient has presented a great variety of curious symptoms, the analysis of which will be left to another time. In general, it might be said that the following groups of symptoms lasting for weeks or months might be of interest from the point of view of this paper. For periods of a few weeks to a few months the patient has presented the interesting phenomenon of a presumably involuntary paralysis of all the extrinsic muscles of the eyeball. This was at times accompanied by a sort of spasmodic contraction of the muscles of the jaw which prevented him from opening his mouth except for a very small space, only sufficient to admit liquid food. There developed at times a double-sided ptosis, which disappeared without leaving any trace. There was also a temporary paralysis of the mechanism of deglutition, which necessitated rectal feeding for a time. About two years ago developed a peculiar series of automatic movements, especially marked in the arms, though present to some extent in the lower extremities. These movements were to a certain degree under the control of the will, but their full performance was subject to such variations of control that as a rule the movements were overacted; that is, they were not finely coördinated, so that there was a peculiar jerkiness to them that resembled to a certain extent tabetic movements. These are only a few of the more important curious phenomena which this case has presented from time to time.

It should be noted that during all this time there was no appreciable mental change. The patient to-day retains full vigor of intellect and employs himself in reading history, travels, philosophy and other books of a like nature; in fact, it is interesting to note that mentally he appears to have deepened his powers of thought, especially in directions of analysis. His disinclination to talk, much increased of late, has prevented an insight into the deeper psychology of many of these phenomena which his growth in analytic power might have made deeply interesting.

This, in brief, is an outline of the case, and any explanation which might be made must be based upon as clear a conception as is possible to obtain of the state of mind which the patient has presented to himself. The evidence of Dr. G—— himself forms a body of observations which must be acknowledged are worthy of respect and should be the groundwork of any hypothesis which we advance. The main fact to be obtained in this way is the statement that in no part of his life has he felt that his power has ever been equal to the necessary performance which has been imposed upon him. From boyhood to the present time this uncertainty has been one of the accepted facts of his life probably the most apparent. So long as this had to do purely with the activities of a physical nature, he was enabled to get through them in some way or another. At the point when the purely muscular functions became involved as a result possibly of the influenza attack there developed the paralysis and the purely motor phenomena. If we grant that this is a reasonable explanation of the origin of the paralysis, then we can easily see why it is that this state has persisted and will likely persist as long as the patient lives. There is no doubt in the patient's mind, and there never has been, of his ability to move his muscles to walk, if necessary; to rise from his chair if the occasion required. The point is that any such effort would appear to him a useless exercise and a futile using up of what volitional power there remained left to him. He has made for himself a material conception of the amount of voluntary power which he as an individual possesses, and this amount he prefers to leave undiminished. There exists for him no obvious reason why he should change his present condition for one which would awaken in him the old struggle against doubt and uncertainty. This is the point, to my mind, which differentiates this case immediately from hysteria. The attitude of this patient's mind is and always has been perfectly conscious. As far as I have been able to ascertain, there has never been a symptom which the patient did not perfectly understand, as far as his understanding went. Hysterical phenomena are most easily appreciated if we assume that their origin at least is purely sub-conscious. We are all ready to acknowledge, I believe, that an hysterical patient is unaware of the anomalies which he presents until his attention is directed to them by examination or observation. In this case, however, the symptoms have been a result, we might say, of an almost conscious adaptation to the patient's assumed inability for any sort of an active life. In this statement the obsession of the fixed idea comes to the front, and the basis of the contention that this is a case of psychasthenia might be said to rest here. A patient with his psychical self so deeply pinned down to a sense of his own powerlessness, thus giving way to the morbid introspectiveness markedly present from the start, might very readily develop any symptom or group of symptoms which this patient has presented.

For Janet, doubt, indecision, a consciousness of incapacity, are typical traits of the psychasthenic's mental make-up. In this sense, at any rate, this patient is a psychasthenic. In thinking over this case I have often thought that the term psychical paralysis might adequately describe the condition. The objection to this is that paralysis always assumes a motor function, and here we have a state of lowered activity of the mind itself. The term paralysis is open to the same objection, and in addition this term is already in use for an entirely different symptom-complex. So, after all, the designation psychasthenia leaves us more satisfied than any other. If this exaggerated case can be so described and can be justly set down as belonging to a neurosis that is neither hysteria nor neurasthenia, then there is a wide field for the investigation of milder cases for which there has, up to this time, been no satisfying explanation.

In conclusion it might be a point of interest to note that the patient himself has often stated that he looked upon himself as a sort of Hamlet, and the more one thinks of this the more one sees the justice of the comparison.

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

April 4, 1905.

The President, DR. JOSEPH FRAENKEL, in the Chair.

Trophic Disorders in Diseases of the Nervous System.—By Dr. William B. Noyes. The speaker said that many of the most interesting and least understood subjects in medicine could not be claimed exclusively by any one specialty, and the purpose of his paper was to group together cases where changes in the nutrition of the tissues were found closely associated with lesions of the central or peripheral nervous system. In a few instances this connection would be found to be positive; in others it would be little more than a hypothesis. The practical result of the study would be to turn the attention of the physician treating a seemingly local disease of the skin, joints or other soft parts to a broader view of the case, even if it should bring him face to face with some disorders of the nervous system more difficult to handle than the superficial lesions alone.

The skin, the most obvious field for clinical observation, showed most clearly and promptly any change in the general bodily nutrition. The epidermis, hair or nails might each or all be affected. The hair turned gray or white, became brittle or fell out in conditions of failing nutrition, and the changes physiological in old age, might appear in a few hours in cases of great mental or nervous shock. General or local alopecia were in many cases directly associated with functional or organic disorders of the central nervous system. A neuralgia might leave as a permanent mark a patch of gray or white hair. The nails might undergo rapid or gradual change, especially noticeable in multiple neuritis. The skin might suffer atrophy of the cutis, epidermis or both. Senile atrophy of the skin, associated with increased pigmentation of the epidermis and thinning of certain layers, was a condition which might under certain circumstances be seen earlier in life. An idiopathic diffuse atrophy might develop, with general thinning of the integument, increased prominence of the blood vessels and a characteristic red color. After division or destruction of a peripheral nerve by disease certain well-defined trophic disturbances of the skin would occur in time. The skin might also suffer from hypertrophic processes, with hyperplasia affecting its different elements and the subcutaneous tissues. Of this, an ordinary corn was the most simple example. This might become actively inflamed and develop into an abscess, with the subsequent formation of a perforating ulcer. Such perforating ulcers, though appearing in various nervous diseases, were usually associated with locomotor ataxia. Gasquill, who had carefully analyzed 83 cases, found 32 in tabes, 17 in general paralysis of the insane, 14 in diabetes, 8 in alcoholism, 12 in various lesions of the spinal cord, and four of traumatic origin. It also occurred in anesthetic leprosy and syringomyelia. Another disease illustrating hypertrophic changes in the skin was sclerodema, the lesions being characterized by a general induration and immobility of the affected tissues. Nearly all acute skin diseases were vascular disturbances. Anemia of the skin might be general or localized: if the latter, it was due to pressure, constriction or spasm of the vessels, and was therefore a vasomotor neurosis. Chronic ergotism would produce similar paralytic conditions of the vessels, with formication, pain, spasm, anesthesia and coldness, and later local gangrene. The extreme type of this condition was seen in Raynaud's disease, a rare trophic disorder, where the vasomotor spasm developed (1) local asphyxia; (2) symmetrical gangrene.

The skin diseases whose lesions showed a passive or active hyperemia, including the erythemas and dermatites, embrace few examples where any definite nerve lesion was present. A familiar example might be mentioned in the ordinary chilblains, which were the result of a vasomotor neurosis due to vasomotor paralysis from cold. Erythromelalgia, which all modern text-books on nervous diseases classed as a clinical entity, was a rare disease, with exaggerated vasomotor changes, associated with burning sensations, pain, redness and localized swelling. Chronic urticaria was said by as high an authority as Unna to indicate some alteration in the nerves of the vessels. Angioneurotic edema, closely related to urticaria, though originating in the subcutaneous tissues, had recently figured extensively in neurological debates. It stood in the same relation to the central nervous system as exophthalmic goitre, namely, through the sympathetic nervous system.

Herpes zoster was most instructive in showing the relation between the skin and the central nervous system. All recent authorities agreed that it was secondary to changes in the nervous system, and was associated with an acute neuritis. This neuritis was dependent upon lesions of the ganglion cells of the posterior roots of the cord. Another disease similar to zoster was pemphigus. Here again organic changes in the spinal cord and peripheral nerves were reported by occasional observers, but these were not as uniform in location as in zoster, and moreover, these nerve lesions were frequently not found at autopsy.

Psoriasis was one of the most frequent and well-known of skin diseases. That it had a close relation with the nervous system was not commonly understood by dermatologists, but this close relation was maintained by many authorities. It was more often associated with neurasthenia, hysteria, epilepsy, insomnia and other functional nervous diseases than with definite organic disease of the nervous system.

For the proper nutrition of the skin, muscle, bone or other tissue the integrity of the trophic center, its peripheral path and its termination were essential. Any localized and sudden disturbance of nutrition, when not due to shutting off of the blood-supply, was the result of irritation or destruction of peripheral nerve trunks or spinal centers. The nerve endings, if they had not a structural continuity with the tissues, at any rate seemed to modify the nutrition of their cells. It was known that disease of the neurones, whether intraneural or extraneural in its origin, progressed from the center down the neurone or axis cylinder to the end organs. Then, some irritation or failure in vitality appeared in the tissues, be they skin, muscle, cartilage or bone.

In the concluding portion of his paper Dr. Noyes discussed the various trophoneuroses, particularly in their relation to diseases of the joints. Chronic arthritis appeared as (1) chronic serous arthritis; (2) chronic purulent arthritis; and (3) dry, ulcerative arthritis. It was the last form that especially interested the neurologist. It was a local disease of the joints characterized by erosion of the cartilages, thickening of the capsule and denudation of bone. It might appear as a disease of old age, or be associated with organic nervous disease, as in Charcot's joints, or associated with chronic rheumatic disease, or as a chronic arthritis deformans. General paralysis of the insane caused changes of the spinal cord almost identical with those of tabes, and sometimes caused Charcot's joints. Another type of Charcot's joints was seen in syringomyelia, and still another in leprosy. Dr. Noyes then described these different types of chronic ulcerative arthritis, and showed a number of X-ray photographs illustrating the same.

Dr. Henry Rafel said he had frequently heard the statement that persons with psoriasis were otherwise unusually healthy, and this was in accord with his own experience.

Dr. Joseph Fraenkel said that many of the points brought up for discussion in Dr. Noyes' paper rested largely on a clinical basis, and it remained for the neurologists to explain the connection between the various lesions of the skin, joints, etc., and the nervous system. A few of the facts in connection with the exhaustive study made by Foerster in a large number of cases of tabes, which seemed to prove that the sensory neurone, in addition to the conduction of sensation, was a very important factor in the nutrition of tissues.

Two Cases of Acute Homonymous Hemianopsia.—Reported by Dr. B. Sachs.

Case I.—The patient was a man, sixty-three years old, an upholsterer, who had been married thirty years. His family history was negative. The patient had suffered from mid-winter cough, with some dyspnea on exertion, for many years. Eighteen months ago he had slipped, striking his head; he was dazed for a time, but was able to get up without aid and walk home. His urine was normal.. For some time he had noticed an impairment of his sight, particularly at night, when he had to give up reading.

The patient was admitted to Mt. Sinai Hospital on March 4, 1905. Four weeks previous to that time he had suddenly noticed that he could not see an object approaching him on the left side. Pupillary reflexes were normal. There was no double vision, nor other visual symptoms; no headache nor vomiting; no hemianesthesia; no ataxia; no evidence of hemiplegia. Examination showed nothing but a left homonymous hemianopsia, the outer field of the left eye and the inner field of the right eye being obscured.

The interesting point about the case, Dr. Sachs said, was that the condition occurred in a man of 63, of infantile physique, and was apparently due to a thrombosis of one of the branches of the posterior cerebral artery, and there was an absence of the usual symptoms following a thrombosis of one of the branches of the middle cerebral. As to the site of the lesion, in the absence of hemianesthesia and hemiplegia, it must have either involved the optic radiations, or possibly the visual center in the occipital lobe, the only symptom being an hemianopsia, and without local pain. The speaker said he was inclined to believe that it was due to an area of softening involving the optic radiations.

Case II.—The patient was a young woman of eighteen. The history she gave was that she had been suffering from a slight nasal catarrh, and a few days ago, on blowing her nose, she was suddenly seized with an intense headache, and a feeling as though something had burst in the back of her head on the right side. She also immediately noticed that something was wrong with her. vision. The only symptom her physician could find was a left lateral hemianopsia, and this diagnosis had been made also by Dr. Charles L. Dana, who had previously seen the patient.

As to the cause of the lesion in this case, the diagnosis rested between hemorrhage and an embolism. The girl had a distinct cardiac murmur and a dilated right heart, probably the remains of an old endocarditis. The speaker said he was inclined to believe that with an embolism the general shock would have been greater than it was. On the other hand, the intensity of the headache, and the rapidity with which the symptoms developed, seemed to point to a hemorrhage as the cause of the trouble, in spite of the doubtful heart condition.

A Case of Diplopia.—Reported by Dr. Sachs. The patient was a man, thirty-five years old, a resident of the South, who on November 1, 1904, was thrown from his buggy, striking on his head. He was unconscious for twelve days after the accident. When he regained consciousness the physician who attended him noticed a slight difficulty of speech, and a little stiffness when he attempted to walk, but there was no paralysis.

When Dr. Sachs first saw the patient, about two weeks ago, the only symptom he could find was a peculiar form of diplopia, apparently resulting from a paralysis of the left superior oblique muscle. This was confirmed by Dr. A. Wiener, the oculist. This muscle was deficient, and gave rise to two vertical images.

The case was interesting because of the comparatively slight residue of what was apparently a severe traumatic hemorrhage in the vicinity of the fourth nerve nuclei

Dr. Arthur C. Brush said that cases of hemianopsia like those reported by Dr. Sachs were not particularly rare. In one case that came under his observation the patient was a young man, an athlete, with a much hypertrophied heart and thickened arteries, who at the end of a bicycle ride from Coney Island to Brooklyn suddenly felt dizzy and could not see. When Dr. Brush saw him on the following day there was a distinct hemianopsia, and nothing else. The man was totally blind on one side of each retina. His only other symptom consisted of severe mental depression. The hemianopsia was permanent.

Another case seen by Dr. Brush was that of a young woman of thirty, who stated that on attempting to lift some heavy weight she suddenly became dizzy, and had also vomiting and headache, and partial loss of vision on one side. This on examination proved to be due to a hemianopsia. In both of these cases there was evidently a predisposition to the occurrence of arterial lesions. Where the particular lesions were in these cases the speaker said he did not know.

Dr. William M. Leszynsky said that during the last four or five years he had seen several cases of complete homonymous hemianopsia at the Manhattan Eye and Ear Hospital. In the absence of evidence of involvement of any other portion of the optic tract, he was forced to conclude that the lesion was situated in the cuneus, being due to hemorrhage or thrombotic softening. All the patients were about fifty years of age.

Dr. J. Ramsay Hunt said that last summer he saw a woman, about sixty, who suddenly developed an isolated paralysis of the right superior oblique muscle. She was examined by Dr. Colman W. Cutler, and showed the characteristic diplopia. There were, however, no other evidences of acute nervous disease. On account of her age, a vascular lesion in the bulb or pons might be suspected, but the paralysis cleared up entirely within two weeks. Before going to bed she had washed her hair, and the next morning the impairment of vision was noted, accompanied by a neuralgic pain in the orbit, and a tendency to dizziness. There was no vomiting. In the experience of ophthalmologists, Dr. Hunt said, these cases are not extremely rare, and they interpret them as rheumatic neuritis of the trochlearis nerve.

Dr. Hunt said he recalled a case in which the hemianopsia was complete, and was evidently syphilitic in origin. In another case, a man rather advanced in life, the hemianopsia appeared suddenly with vertigo and headache. In such a case the lesion is probably situated in the occipital branch of the posterior cerebral artery.

Dr. Leszynsky said that paralysis of the superior oblique was not uncommon, and when it did occur it was often difficult to tell whether the diplopia was due to paralysis of the superior oblique or of the inferior rectus. The correct interpretation of the condition required a long and careful study of the false image associated with the diplopia. The speaker said he had never seen the superior oblique involved simply as a residual symptom.

Dr. Sachs, in closing, said he did not wish to be understood as saying that he regarded hemianopsia as a very rare condition. In both of the cases he had reported the heminaopsia came on in a very acute fashion, and was unaccompanied by other symptoms. Of course, the symptom was not uncommon in cases of cerebral paralysis. The speaker said he had never seen a paralysis of the superior oblique as the sole residuum of a rather severe injury, such as that reported in his first case.

CHICAGO NEUROLOGICAL SOCIETY.

Thursday, April 20.

The President, Dr. H. N. Moyer, in the Chair.

A Case for Diagnosis (Multiple Sclerosis?).—This was presented by Dr. D. O. Hecht at the request of Dr. Patrick. D. E., a male, forty-seven years old, of Irish and Welsh descent but born in the United States, was seen for the first time in March, 1905, at the Clinic for Nervous Diseases, Northwestern University Medical school. He is a butcher by trade, has been married twenty-two years, and is the father of thirteen children. Eight of the children are living and enjoying good health. The family history is negative. The period of infancv and early childhood was uneventful.

At the age of twenty-one the patient came to Chicago and got employment as laborer in the Stock Yards. During his early experience there, which covered a period of four or five years, he was compelled to do various kinds of work, some of which took him into cool cellars for days and months at a time; but it was before the era of ice machines, and the cellars were not nearly so cold as they are now.

In 1889 (sixteen years ago) the patient experienced for the first time dull. boring pains in the muscles of the small of the back, which came and went at close intervals, and were especially keen upon efforts at bending far forward and backward or straightening up from a stooping posture. His description of the discomfort reminds one of the lumbago pains. The shoulder, elbow and knee joints have been the seat of localized pain, severe enough at times to be quite distressing, and on several occasions the fifth and sixth intercostal areas on the left side have been invaded. Inquiry reveals that there has been little or no stiffness in the parts affected, but for the most part just enough soreness to interfere at times with work. The painfulness at the elbows often made it very hard for him to hang sheep on hooks preparatory to skinning them. Wet and damp weather were without influence; long sitting or inactivity did not stiffen the parts involved; swelling and other periarticular symptoms were absent.

Up to the time of the patient's coming to Chicago to work in the "yards" he had been totally abstemious in regard to drink, and a man of modest habits in all things; with his new occupation he took to drinking both whiskey and beer in quantities, and its was not long before he was consuming six or more whiskies a day and as many as ten pails of beer. The practice of this excess was kept up for fifteen years, and was brought to an abrupt end in January, 1905. He ascribed his hard drinking to the fact that it did not affect him as it did other men; only occasionally had he been intoxicated; his head may have suffered, his legs never.

Onset of the present illness was about six years ago, with a sense of weakness and "giving away" in the knees. This was especially felt while carrying sheep into cellars after they had been killed and skinned. The feeling of uncertainty and weakness increased to a point where, after the first year or so, he noticed it when walking on the level. Attributing it entirely to his overindulgence in liquor, he thought it unnecessary to consult a physician, and the fact of his not losing a day from work strengthened the belief that his was but a transitory difficulty. In the act of stabbing and skinning sheep it was customary to hold them fast between the knees. The strain of this position on the increasingly weak leg adductors, together with self-inflicted cuts on the hands from repeated slips of the knife, caused him enough discomfort and concern to seek medical advice.

About a year ago his hands, especially the left, began to show signs of shaking, which became so aggravated that drinking from a cup held in the left hand was attended with great difficulty. To a less degree the right hand was in the same plight. In his opinion, the crude strength of his hands is not what it was, but it is infinitely better than that of the legs, which have become progressively weaker, until now he regards them unequal to the least sustained strain. Neither rigidity nor dizziness is complained of. At no time during the past fifteen years has he experienced root pains or sensations of numbness, deadness or tingling anywhere in the body.

Sensory disturbances, both subjective and objective, have always been, and are at the present time, conspicuously absent. About six months ago the patient found it necessary to wear glasses for reading purposes; vision for distance remains very acute. Hearing and the senses of taste and smell are in no way impaired. There are no bulbar symptoms. There has been no complaint of vesical or rectal disturbance of any kind. The bowels move daily; the appetite is fair; the sleep is good, and there has been no appreciable loss in weight.

Status præsens: The patient is a tall, well-developed man and weighs about 175 pounds. Examination of the thoracic and abdominal viscera proved negative. There was no temperature; the pulse count was 80. The blood and urine findings of a single examination were negative. The special senses show nothing of interest. There is no anomaly of speech. The cranial nerves are nowhere involved, and the fundi oculi reveal no changes. There is no nystagmus. The entire clinical picture is evolved from motor premises, and the gait immediately attracts attention. It impresses one as spastic, wabbly and uncertain, as if the knees were giving away and unable to keep the feet in the line of direction. The eyes are held riveted to the floor, as is the habit in tabes, and the heels, which are brought to the floor first, come down hard enough to jar the whole body. The shakiness in the limbs is more noticeable to the patient in turn-about movements, and when he first gets up from a chair and starts off. The Romberg sign is not present.

In the recumbent posture the inco-ordination of the lower limbs appears still more marked. Attempts to fix the right heel to the left knee are not successful, and the same holds good in fixing the left heel to the right knee. The excursion to the knee is good, but a violent jerking and floundering about attends the effort to hold it fixed. After repeated trials some of the unsteadiness is overcome and fixation meets with partial success. Control in this maneuver is also better with the clothes and shoes on, since their weight is prone to limit the irregular jerking movements.

With the patient on his back, the legs raised and spread widely apart, the limbs and trunk oscillate in all directions in a most disorderly fashion, despite straining to control same. It is impossible to maintain fixation for more than a few seconds, and the arythmic movements are more aggravated with the whole leg extended on thigh in midair than with the upper leg extended on thigh and lower leg flexed from the knee. There is an increase in the amplitude of motion when the legs are widely adducted, and a decrease when brought in apposition. Co-ordinate movements of the arms and hands are much better executed than those of the lower limbs. At times he can pick up a match or pin from the floor with ease, but says this power varies and it is at times very hard to do. Pouring water from one test tube to another reveals a coarse tremor. The right index finger is brought more steadily and directly to the nose than the left. He approximates the indices fairly well, but strangely enough, he does this maneuver much better standing with the feet closely together (as in Romberg position) than with feet spread. Large movements of the arms accompanied by notable lack of balance in the trunk and legs.

Protrusion of the hands and separation of the fingers reveals, more in the left than the right, coarse, irregular movements. Refined tests applied to the smaller musculature, such as needling and buttoning, elicit marked tremulousness. The crude muscular strength, when measured by the examiners, is shown to be not much impaired. He overcomes tests of antagonism with good strength. Adductor and abductor groups in the legs seem stronger than the patient is willing to confess, and the same holds good of the flexors and extensors. In testing the lower limbs together, the left side may have been a shade weaker than the right. The left grip appears slightly weaker than the right, but this is also relative. There is no visible atrophy, but here and there the muscles feel a bit flabby.

The pupillary reflex is normal to light and accommodation; the jaw-jerk was not elicited. With the arm hanging loosely over the back of a chair, the triceps-jerk seemed more responsive on the right than on the left. Wrist taps were not elicited. The pectorales and spinati show excessive myotatic irritability and exaggerated responses. The knee-jerks were bilaterally exaggerated. Abdominal skin reflexes were present, but the cremasteric was not elicited. The Achilles-jerks are bilaterally equally brisk. The Babinski, Gordon and Oppenheim reflexes were absent. No ankle clonus; plantar skin reflex was very sensitive. Fibrillary twitchings were absent. The tactile, pain and thermal senses are perfectly intact and exquisitely accurate. The sense of orientation was exceedingly good, even when confined to slight degrees and angles of deflection in the single toes.

Dr. Hecht said that when he first saw this case, in the absence of any sensory phenomena and in the presence of spasticity and what he took to be inco-ordination and not tremor, he thought of its being perhaps a primary spasticity, and the lesion limited to the lateral tracts. That view would not explain the inco-ordination, the ataxia the man had. On walking, the patient showed the jerking of multiple sclerosis, the quivering and shaking throughout the whole body. The head moves a little. There is no nystagmus, no scanning speech—nothing more than this fair degree of spasticity.

Dr. Patrick said he thought the condition was typical of multiple sclerosis. Dr. Hecht had been under the impression that the trouble would show more in the excursion than in the fixation. Dr. Patrick spoke of the condition in many cases showing in the act of drinking; the patient brings the glass up fairly well, and then the trouble begins.

Dr. Hecht said he thought this was a tremor of multiple sclerosis, and considered the inability to fix as an ataxy. The man has been sick six years, and there have been remissions, the only history being that of pretty strong alcoholism.

Dr. Kuh said that against disseminated sclerosis was the late onset. It never begins so late as in this case. He thought, however, disseminated sclerosis, in the broader sense, was pretty safe.

Dr. Williams said it was hardly diffuse sclerosis causing a combined system disease, as in six years it would not fail to have shown some symptoms. He had recently read an article in which a systematic test had been made of the bone sense, and it was found the only sensory symptom present in tabes, for example, and in mixed sclerosis. Diabetes was the only other condition in which it was present in this series of 200 cases, and never present in condition of health.

Dr. Kuh said that in the majority of cases it was not usual for the tremor to be greater in the legs than in the arms in multiple sclerosis, but there was no reason why it should not be. The lesions are more marked below the cervical margin.

Dr. Patrick said that he thought the reason the intention tremor in the legs is not more frequently mentioned is because it is not frequently examined for. He frequently finds it.

Dr. Hecht said he thought the text-books were somewhat in error in giving the impression in regard to the tremor, that the excursion was very good, or rather, that in the effort to fixate, it subsides.

Dr. Kuh said the reference was made to the effort on the part of the patient. In bringing the fingers together they travel pretty well, and at the stopping place the trouble begins. Asking the patient to bring the fingers together and stop just before touching and hold them there will often bring out the intention tremor when the ordinary test will not.

Sarcoma of the Brain, with Development of Acute Symptoms Two Weeks After an Adenoid Operation.—This case was presented by Dr. J. H. Hess ,who first described the tumor and showed the specimen. The child (five and one-half years of age) had received a blow on the head a year and a half ago, but on examining the skull there were no findings in that region. The dura was very deeply injected all around, but there were no other changes except at the base, where there was a slight meningitis. In no part of the dura were tubercles visible. The pia presented about the same condition. Upon external examination there were no visible changes of the brain. On section and opening into the lateral ventricles a large, mulberry-like mass was noted in the anterior horn of the left lateral ventricle, which goes through the corpus callosum into the anterior horn of the right lateral ventricle, the mass being about the size of a duck's egg, reddish brown in appearance and containing no nodules. The posterior horn of the right lateral ventricle was filled with a similar mass. The choroid plexuses on both sides were normal. There was a very marked internal hydrocephalus. In the tumor mass of the posterior horn of the right lateral ventricle was a small abscess containing sero-purulent matter, and anterior to this were four small cysts containing clear fluid. The right ventricle was filled with a sero-purulent fluid; the left with a clear fluid. The anterior half of the right lobe of the cerebellum was composed of a tumor mass. Microscopic examination showed a small round-cell sarcoma.

The history is even more interesting, and follows: Clara S., aged five years, one and one-half years ago received a blow on the head with a brick one-half inch to the left of the median line of the forehead at the hair line. The wound bled profusely and caused headache for ten days, which disappeared and she regained her usual good health. One year ago she complained of earache, which was followed by a purulent discharge from the same ear, the side not being remembered by the mother. Six months ago she suffered from recurrent headaches, which the mother described as "spasmodic flashes," coming on suddenly with flushing of face, severe pain, and shortly disappearing. These continued more or less irregularly up to October 19, 1904, when both tonsils and a large mass of adenoid vegetations were removed. Two weeks later she complained of severe headaches and dizziness, followed shortly by severe attacks of psychic vomiting, apparently without cause and without definite relation to the time of eating. She also suffered at this time from vertigo and internal strabismus of right eye, which cleared up in three days. During this time the temperature ranged between 98.6 and 103 degrees F. Pulse is not remembered. She also complained of severe frontal headaches and pain in the right shoulder and arm, apparently due to nerves of brachial plexus. Flexion and extension of head at this time produced no symptoms.

These symptoms improved, with irregular exacerbations, and three weeks later she developed what appeared to be a spasmodic torticollis. Still later, when this disappeared she had photophobia, and the mother noticed ataxia in gait. Since then the symptoms have persisted, with improvement at times. The mother also noticed limpness in the left arm, which improved, but did not entirely disappear. Headaches appeared suddenly, which, at rest, disappeared as suddenly. While out walking she suddenly complained of blackness before the eyes and had to be carried home.

March 10, 1905, 10 A.M., when seen she had right-sided facial paralysis, with paresis of left leg and weakness of left arm, although able to grasp the hand with considerable vigor. The mind was clear, the little patient remembering the doctor's name, from a visit two months previously. The mouth was drawn to the left in speaking, and the right eyelids did not close. Photophobia was present. Tongue was slightly drawn to the left, and she could not make grimaces with left side of the face. Slight strabismus of right eye had apparently not improved. Some retraction of head was present, also severe frontal headaches and drawing up of legs; pulse 80. Pupils react to light sluggishly; abdomen retracted, reflexes absent.

At 6 P.M. of the same day opisthotonus; legs flexed on abdomen and child comatose. Temperature 98.8, pulse 108.

March 11th, 10 A.M., temperature 100.6; pulse 108. Opisthotonus; legs flexed and rigid; child comatose; pupils reacted, left fairly well, right sluggishly; choked disc. Bowels moved involuntarily first time; spine apparently very sensitive in cervical region; knee reflexes absent.

An interesting feature of the case is that the child had very few symptoms before the adenoid operation, but it was unfortunate that she began to have many symptoms two weeks following it, leading to the supposition that the operation may have been a direct etiological factor in her illness. This, however, was disproved at the autopsy. The test tubes were found to be infected, and it was a question whether the fluid was pus or necrotic tissue.

Dr. Harold N. Moyer said that the case illustrates several things apparent in tumors in children; he had been struck by the enormous size of tumors in children. These are probably never diagnosticated until late in the disease. He had a case that resembled somewhat the one related by Dr. Hess: the same obscure and scattered symptoms, choked disc, the patient was taken with sudden blindness, and died three or four days afterwards. The child had cerebral vomiting and all the more distinct signs of cerebral pressure. The very large tumor mass in this case is also significant of the tumors of children. The skull is flexible and tolerates pressure, and the tumors are larger than in adults.

Dr. L. H. Mettler, in connection with this, called attention again to the case of the nine-year-old boy shown at the last meeting, which he had very hurriedly and inadvertently brought in as hysterical amblyopia. The Saturday after that meeting the child was examined by Dr. Beard very carefully as to the eyes. The fundi, at a casual examination, would be considered normal; absolutely no atrophy. Around the maculæ there was a little change in the blood vessels, a slight edema; the more prominent vessels showed a slight shadow. The color had faded somewhat, and there seemed to be a slight extravasation in the minute blood vessels. He said he had occasionally found such conditions where, later on, a neuroretinitis appeared. Dr. Mettler had asked that the boy be brought for complete examination, but the father returned to say that they had decided to trv Christian Science and let medicine alone. Dr. Mettler thought there was no organic growth at the base of the brain.

Dr. Sydney Kuh said he thought it a rule rather than an exception to see a post-mortem rise in temperature in death from brain tumor. He had, in a recent case at the County Hospital, asked that the temperature be taken after death, and the rise was noted. In Heidelberg they had always looked for it, and frequently also in meningitis cases. It often is 104-5-6 or 7 degrees half an hour after death.

Dr. Hess said that the child's mind was very bright. She was rational and her memory was perfect, which in view of the callosal invasion, was remarkable.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

April 20, 1905.

Dr. P. C. Knapp in the Chair.

A Case of Hemiplegia with Peripheral Paralysis of the Seventh Nerve on the Same Side.—This was shown by Dr. Courtney. This young woman —now twenty-five years of age—at the age of eight had what was probably a virulent form of scarlatina, complicated by encephalitis and purulent otitis media. As a result of the encephalitis she has a very spastic hemiplegia of the right side, with intensely rigid contractures at wrist and ankle; and as a result of the middle ear trouble, she has, in addition, a profound peripheral facial palsy, also on the right, with degenerative changes in the affected nerve.

The interest of the case lies in the fact that Nature has been doubly cruel in this instance, in the implantation of an incoercible peripheral palsy upon an unusually severe palsy of central origin.

A Case of Purulent Otitic Meningitis.—This was reported by Dr. Knapp. Dr. Knapp was called to the hospital by Dr. Leland on the evening of April 18th to see a woman of forty-six, with the following history: She had suffered with pain in the right ear for three weeks, which was not relieved by treatment, and which later shifted to the temples. Three days before, she went to her physician's office, still complaining of pain, and was given sedatives. The night of the 17th she became unconscious, and the next day was brought to the hospital. When Dr. Knapp saw her she was unconscious, but moaned when moved or handled. The temperature was 102.6, the pulse 110 and very weak. The temperature fell to 101.5 after a bath, but rose again. On entrance she did not respond promptly when the right side was pinched or pricked, but she did when the left side was stimulated. There was no retraction of the head, but decided movements of the head caused louder moans. The right pupil was twice the size of the left, but both reacted to light. The right eye was not turned outward beyond the median line, and the left was apparently not turned outward fully. There was a tendency to ptosis in the left eye, and to turning of the eye down and in. The ophthalmoscope showed nothing remarkable in the right eye, but it was impossible to get a clear view of the left fundus, owing to the movements of the eye. The mouth was drawn to the left, but the right eyebrow was occasionally raised. When Dr. Knapp saw her she moaned and withdrew both limbs equally when pricked on either side. Nothing abnormal was found in the chest or abdomen.

The knee-jerks were lost. On testing the plantar reflexes there was slight flexion of the toes, but a very lively purposive withdrawal of the foot. The absence of pressure signs, the involvement of the cranial nerves, and the high temperatrue, led Dr. Knapp to suspect meningitis rather than abscess, and that opinion was confirmed by the absence of pressure revealed when Dr. Leland exposed the dura, so that Dr. Knapp advised against further exploration of the brain. The breathing had approached the Cheyne-Stokes type before the operation, and assumed that type decidedly afterwards. The patient died nine hours after the operation.

Dr. G. A. Leland said that the condition of the ears in this patient was obscure, and one that would probably be easily overlooked by the general practitioner. In fact, the diagnosis on entrance was abscess, or tumor, of the brain. There was no redness nor swelling of the tissues over the mastoid process, only doubtful tenderness on very deep pressure. Examination of the drum-head, however, showed redness extending to the periphery and contiguous parts of the canal, but no bulging nor swelling. The drum-head, however, was one which showed previous inflammation, being thickened and scarred, and evidently with adhesions to the promontory, which accounted for its nearly normal position, a drum-head which does not yield to internal pressure, and therefore allows of inflammatory exudate without the ordinary objective appearances; and it is to be inferred also that inflammatory products will probably go inward rather than come outward.

On opening the mastoid it was found about evenly divided between pneumatic and diploetic spaces, the former being filled with thickened edematous, spongy granulations; but there were nowhere collections of pus, the bone showing intense inflammation throughout. The mastoid process was entirely removed by operation, and the dura exposed in the middle fossa through the tegmen for a considerable area, and also in the peri-sinus groove. The dura in the middle fossa was smooth, rather pale gray in color, not bulging into the aperture and not pulsating. The dura in the sinus groove was intensely red, not smooth and shiny as it should be, but easily bleeding. The sinus itself was soft, showing evidence of thrombosis and only respiratory pulsations.

It would seem, perhaps, that an operation in this case was not justifiable when the findings were considered, but the cranial cavity was opened after obliterating the focus of inflammation, because of the results obtained in two cases operated upon under similar conditions, where the diagnosis was meningitis, where the duration in one case was much longer, and where both cases cleared up and were discharged well. White count showed 43,000 before operation and 50,000 the next morning.

The post-mortem examination was reported by Dr. S. B. Wolbach: On opening the dura there was no evidence of pressure. Adherent thrombi existed in the left lateral sinus near the torcular Herophili and close to the jugular foramen, grayish pink and friable.

Along the course of the vessels of both hemispheres was a greenish-yellow exudate, subpial, extending to the median fissure. The base of the brain, the cerebellum, was covered by a similar exudate, specially thick about the third nerve. The exudate extended downward from the pons, medulla and cord as far as could be seen through the foramen magnum. In each ventricle was yellowish, cloudy liquid.

The right middle ear and antrum showed a destructive purulent process. Cover glass preparation from the exudate from the base of the brain and ventricle showed many lanceolated gram-staining diplococci.

The anatomical diagnoses were: Pneumococcus meningitis; thrombosis of left lateral sinus; chronic otitis media; infection of sphenoidal sinus; mastoid operation.

Arteriosclerotic Brain Diseases.—Dr. Albert M. Barrett read the paper of the evening on this subject.

The better understanding of the pathological anatomy of general paralysis and the importance which the plasma cell infiltration of the vessel lymph spaces is known to play has cleared the way for the separation of the arteriosclerotic diseases of the cortex. In the same way the recognition of senile brain atrophy without arteriosclerosis shows an independence of two processes often associated in the brain in the later years of life. Arteriosclerosis of the brain must be considered as two processes, viz., that present in the vessels and the reactions in the nervous tissue. The changes in the larger brain vessels are quite the same as in vessels of similar caliber elsewhere. In the finer vessels, especially of the cortex, there occur proliferative changes in the intima which later may undergo various degenerative phenomena; the elastica also participates in these changes. The latter degenerative changes may be of a variety of types; in some instances the altered vessels give the reactions of hyaline or colloid material. The changes in the nervous tissue are pre-eminently of a focal nature. There may be acute softenings where the obstruction of a vessel is sudden or complete, or where the vessel lumen is only partially closed, so that only a lessened amount of nutrition is supplied; there occurs a degradation of the least restive elements, which the nerve cells and fibers, and proliferative glia phenomena in the region of the obstructed vessels. The changes are not specific for arteriosclerosis, yet in their focal nature and their relation to vessel changes are quite characteristic.

The reader reviewed the work of Alzheimer and Binzwanger, and then gave clinical descriptions and the anatomical findings in several cases. There were four cases of arteriosclerotic brain atrophy which clinically closely resembled general paralysis, but anatomically there was present severe arteriosclerosis of the cortical vessels, with multiple focal destructions of nervous tissue; one of these cases had, in addition to the arteriosclerotic symptoms, sensory disturbances with absent tendon reflexes, the anatomical findings in the cortex were the same as in the other arteriosclerotic cases; in the pons were several small cysts among the pyramid fibers, and in the cord there were degenerations in both lateral pyramidal tracts and posterior column degeneration corresponding topographically with that of tabes. The clinical features of the above group were analyzed, and a diagnostic comparison made with general paralysis.

Another group embraced the coarse arteriosclerotic lesions, such as large hemorrhages and softenings; in these cases there is often present more or less severe changes in the cortical vessels with focal destruction of nervous tissue.

Senile dementia is not a pure arteriosclerotic disease, but there are cases of senile dementia in which, in addition to the senile changes, there occur multiple focal devastations in the cortex, and in which the clinical picture shows many symptoms referable to focal changes in the brain.

The reader then gave an analysis of a group of senile arteriosclerotic cases. At the close of the paper a number of slides were projected illustrating the pathological anatomy of the arteriosclerotic disease of the nervous system.

Dr. Walton said that the subject of arteriosclerosis is very important, and one too little considered in neurological study and practice until recent years. Recourse is often had to doubtful etiological factors in case of general and special nervous symptoms for which arteriosclerosis would furnish a simple and adequate explanation. Attention has recently been directed to the many cases of so-called neurasthenia in the aged, in which the diagnosis of arteriosclerosis would be more appropriate. Care must be taken to examine all of the points at which the arteries are palpable before throwing out this diagnosis. Cases occur in which one radial artery is perfectly normal while the other is markedly sclerotic. It is desirable that statistical data be collected bearing on the question of lead in the causation of arterial sclerosis and interstitial nephritis. Dr. Walton had seen both conditions in lead poisoning, but he did not feel absolutely confident that it was more than a coincidence, especially since statistical study has thrown doubt upon alcohol, formerly everywhere regarded as a cause of atheroma, and especially since heredity has been shown to play so prominent a part in arterial degeneration.

Dr. Stedman was interested in the diagnosis of these conditions, and was not surprised to hear that occasionally it was practically impossible to differentiate clinically between cerebral arteriosclerosis and general paresis. Two cases of his showed this similarity, the most recent and striking one being pronounced to be cerebral arteriosclerosis by a number of alienists. It was characterized by moral lapses, gradual mental enfeeblement, shown in increased childishness, pronounced memory-defect and confusion; also frequent congestive attacks with unconsciousness, emotional states, coarse tremors, exaggerated reflexes, unsteady tongue, ataxic symptoms, irregular handwriting with omission of letters and words, and finally complete illegibility, etc., etc.—in short, a train of manifestations so typical of the demented form of general paresis as to lead him to stand out for the latter diagnosis.

Dr. Stedman also thought that we should have to recast our views of the causation of arteriosclerosis in one particular, as recent investigations seem to show quite conclusively that it was not the prominent factor it has been supposed to be. He quoted Dr. Richard Cabot as reporting, among other similar findings, that only 6 per cent. of 283 cases of chronic and excessive alcoholism under fifty years of age and non-syphilitic showed any evidence of arteriosclerosis; that of 45 cases of arteriosclerosis with the same history examined by him only 13 per cent. gave any history of alcoholism: that of 656 autopsy cases of arteriosclerosis only 14.5 per cent were under the age of fifty; and out of the 95 cases showing arteriosclerosis post-mortem but 17 per cent. consumed alcohol to excess. Duclos, who has most exhaustively analyzed these cases, and Ribberts are outspoken in their skepticism regarding alcohol as a common cause of this disease.

Dr. Geo. T. Tuttle said that Dr. Barrett's presentation of the subject is extremely interesting and valuable. He has reported a larger number of cases than Alzheimer had, whose conclusions were based on the observation of only 12 cases when he first wrote on arteriosclerotic dementia in 1895.

The differentiation of arteriosclerotic dementia from ordinary senile dementia, and especially from general paralysis, marks a distinct advance in psychiatry, especially important, of course, with reference to prognosis. It is not so certain that one can always distinguish between the first two, although its occurrence previous to the age of fifty, and the presence of focal symptoms would suggest an arteriosclerotic change. For differential diagnosis between arteriosclerotic dementia and general paralysis, one would depend somewhat on the presence or absence of a previous syphilis; on the age at the time of the attack; on the presence or absence of thickened arteries, enlargement of the heart and kidney symptoms; somewhat, also, on the character of the memory defect, which in arteriosclerotic dementia, as in the ordinary senile form, is liable to be more marked for recent than for earlier events; on the absence of the ordinary physical signs of general paralysis; even after the lapse of several years; and finally, on the preservation of the personality of the individual in the arteriosclerotic dementia, which is very striking; and on a fair degree of appreciation oi the failing memory, and other signs of mental impairment which persists even to an advanced stage.

Dr. Knapp had been impressed with the very early development of marked arteriosclerosis in persons who had led a life of hard physical labor and privation, who were thin and ill-nourished, but who had not had syphilis and had indulged little, if at all, in alcohol. Such cases were met with much oftener in hospital practice, and at a much earlier age, than in alcoholic subjects.

Periscope

Archives de Neurologie

(April, 1905, No. 112.)

1. Note on the Influence on the Offspring of Sexual Excess During Pregnancy. CH. FÉRÉ.
2. Hysteria with Left Hemianesthesia. Alcoholism. Multiple Hallucinations Referred Only to the Affected Side. DUPOUY.
3. Statistics and Instruction of Idiotic and Epileptic Children Living in Insane Asylums. BOURNEVILLE.

1. *Influence of Sexual Excess During Pregnancy.*—Féré's experience has led him to believe that sexual excess during pregnancy is an important factor in the etiology of nervous diseases among children who have an otherwise good family and personal history. He cites a recent case of a boy with sexual stigmata of degeneration who had convulsions during dentation and the eruptive fevers, and at the age of eight developed epilepsy and hallucinations of all the senses. Auditory hallucinations were excited by constipation. The family history was of the best, except for two premature births and one abortion. Sexual relations between the parents were the rule during pregnancy and were infrequent at other times. Féré believes that sexual relations should cease after impregnation, and that a woman's sexual aversion at that time may be the means of protection against premature delivery and malformation, disease and degeneracy of the child.

2. *Hysteria with Left Hemianesthesia.*—Dupouy records an instance of the interaction of the symptoms of hysteria and alcoholism. The patient, a woman of 48, was admitted to the clinic nine times, usually after an alcoholic debauch, and at different times had had left hemianesthesia, temporary hemiplegia, hystero-epilepsy, and hallucinations of sight and hearing, which were referred only to the left side. On the last examination she could barely see, hear, feel, smell or taste with her senses of the left side. The left conjunctival reflex was absent and the left side was weak. At the same time she heard insults and abuses with her left ear, saw terrifying figures with her left eye and had disagreeable hallucinations of smell, taste and feeling. Her left eye was in good condition, and the almost complete contraction of the left field of vision disappeared in a few days under magnetic treatment. Dupouy remarks that the psychoneurosis or hysteria which affected the right side of the brain made this region an area of lessened resistance, and that therefore it alone reacted to the alcoholic poisoning.

3. *Statistics of Idiotic Children.*—As a result of Bourneville's agitation of the subject a commission has been appointed to consider the possibility of applying to children confined in insane asylums the general law of obligatory education for children. Statistics are given which show that in some seventy institutions at the end of 1903 there were 1,206 idiotic and epileptic children, ranging in years from 2 to 18. In six of these institutions schools have been started, and in six others the project is feasible. The author then quotes from nine public or private schools of the sort to show the possibilities. Most of the reports are encouraging. Instruction is given, usually by women, in reading, writing, arithmetic, gymnastics and personal hygiene, and the children's morale is greatly improved. Bourneville urges a campaign for the hospitalization, treatment and education of these unfortunates.

(May, 1905, No. 113.)

1. Hysterical Monocular Amblyopia and Its Disappearance in Binocular Vision. Cruchet.
2. Romantic Self-Identification. Garnier and Dromard.
3. The Psychology of the Degenerates; the Psychology of the Mystics. Binet-Sanglé.

1. *Hysterical Monocular Amblyopia.*—By the study of a girl of fourteen with right hemianesthesia and amblyopia of the same side, Cruchet has been able to confirm his previous conclusions, and further to develop the subject, which is summarized as follows: (1) Hysterical monocular amblyopia disappears at the moment of binocular vision. (2) Binocular vision, however, is not necessary for this phenomenon, as it occurs when the sound eye is shielded. (3) Monocular amblyopia, therefore, arises from closure of the sound eye. (4) This closure acts in a physical way by cutting off the white rays of light.

2. *Romantic Self-Identification.*—The authors describe the case of an impressionable female of thirty, with a neurotic family history, who identified herself with the Claudine of a serial on Claudine at School, Claudine in Paris, etc. She then imagined that her lover was the author, and that her friend was his accomplice in getting the details of her life. She attempted to kill her friend, but her arm suddenly became paralyzed. It is noted that the peculiar development of the retrospective delusions of persecution and the abortive motor reaction show the degeneracy in the case and distinguish it from a chronic persecutory psychosis.

3. *Psychology of Degenerates.*—In this, the introductory lecture, Binet-Sanglé discusses degeneracy as a form of heredity and holds various toxins, etc., especially alcohol, responsible for changes in the germ and sperm cells, which thereupon produce fewer or less well-developed neurones. Then on a biological basis he sketches an evolutionary scale of degenerates from idiots to a high-grade class, who are highly specialized and often very valuable members of the social organism.

The psychology of mystical degenerates is closely related to the psychology of religion. This is a branch of anthropology, and it is to be studied by the usual scientific methods of observation, comparison, generalization and induction. Observation is best employed in connection with historical documents, and the first observations will be on the Jewish prophets.

(June, 1905, No. 114.)

1. The Light Reflex in Paresis. Marandon de Montyel.
2. Foreign Bodies in the Esophagus Among the Insane. Privat de Fortunié.
3. Greffe Thyroidienne (Thyroid Grafting?). Bourneville.

1. *The Light Reflex.*—De Montyel presents a statistical analysis of the light reflexes in 140 paretics which he observed from the beginning of the disease until death. Fifty died in the first stage, thirty-six in the second and fifty-four in the third. The author claims that this is the first complete series of cases, and that the conclusions are more reliable than those based on a much larger number of isolated observations. Every one of fifty-four cases which went through the complete evolution of the disease showed an abnormality at one time or another. In general, during the first stage the number of normal reflexes was nearly as great as the abnormal and alternating reflexes. Among the abnormalities sluggishness and abolition of the reflex in both eyes were frequent and of nearly equal frequency. At the same time there were a few exaggerated reflexes and a few unilateral alterations *en moins*. In the second stage the number of normal reflexes is smaller, and the number of abolished reflexes is greater. In the third stage few normal reflexes remain; the best part were abolished.

2. *Foreign Bodies in the Esophagus.*—De Fortunié mentions two classes of insane who may suffer from foreign bodies in the esophagus: those who swallow such objects on account of their mental condition and those who do so incidentally. In the first class he advocates for diagnosis the X-ray instead of palpation and sounding, and for treatment esophagotomie instead of waiting for spontaneous discharge. He cites two illustrative cases. In the first a woman swallowed her teeth, which were localized by the X-ray near the thorax and extracted by incision of the esophagus. In the second a woman swallowed a stone; the sound passed freely, and no X-ray examination was made. The stomach was opened, but the stone was in the esophagus and was expelled naturally. The second class includes epileptics, idiots, paretics and others who may inadvertently swallow bones or large and hard pieces of food. For them it is necessary to use finely-divided or liquid food.

3. *Greffe Thyroidienne.*—A correction of a typographical error in a review of an article on this subject.

Under necrology appear extensive obituary notices of Dr. Paul Garnier, who died on March 18th. NICHOLS.

Neurologisches Centralblatt.

(Vol. 23, No. 24, December 16, 1904.)

1. The Study in Some Symptoms in Dementia Precox. E. STRANSKY.
2. Nomenclature of "Dementia Sejunctiva." O. GROSS.

1. *Dementia Precox.*—In a long article Stransky details what he considers interesting symptoms of dementia precox.

2. *Dementia Sejunctiva.*—Argumentative article.

(Vol. 23, No. 23, December 1, 1904.)

1. The study of Some Symptoms of Dementia Precox. E. STRANSKY.
2. Heterotopic Innervation. W. S. HUET.
3. Concerning the Combined Occurrence of Myasthenia and Basedow's Disease. R. Meyerstein.

1. *Dementia Precox.*—Continued article.

2. *Heterotopic Innervation.*—Huet records an exceedingly interesting case of a man who, whenever he talked, sang or used his voice in any way, had a fibrillary contraction of the under portion of the trapezius muscle. This association is probably caused by heterotopic innervation of the vagus and the accessory nerves.

3. *Combined Myasthenia and Basedow's Disease.*—The author details an interesting case of myasthenia in which most of the symptoms of Basedow's disease were present. He does not regard this as accidental. and refers to similar instances in the literature. It is possible that both diseases may have a similar etiology.

(Vol. 24, No. 1, January 1, 1905.)

1. Tendon Reflexes and Sensory Disturbances in Tabes Dorsalis. L. BREGMAN.
2. Abdominal Reflex in Tabes Dorsalis. G. CATÒLA.
3. Convergence Cramp in Tabes Dorsalis. H. CURSCHMANN.
4. A Decubitus Lesion of the Penis in Tabes Dorsalis. A. VITEK.
5. The Paralysis-Tabes-Syphilis Question. K. MENDEL.

1. *Tendon Reflexes and Sensory Disturbances.*—Bregman records three cases in which the Achilles jerks were absent while the patellar jerks were still present. He considers the absence of the Achilles jerk one of the earliest symptoms of tabes. He also calls attention to the frequency of sensory disturbances in the distribution of the fifth lumbar and first sacral nerves in the incipient stages.

2. *Abdominal Reflexes.*—The author, to determine the assertion of Sinkler that the abdominal reflexes were exaggerated in early tabes, examined a series of cases and found different results. This sign is variable and not dependable.

3. *Convergence Cramp.*—The author records a very interesting case of tabes dorsalis in which, besides double abducens paralysis, there were nystagmoid movements in extreme lateral directions, and convergence cramp on movement in any direction. Upward converging movement also caused a slight narrowing of the pupils. The cramp of the eye muscles is unusual. It generally occurs in hysteria. There was no doubt of the correctness of the diagnosis in this case. No explanation is given.

4. *Decubitus.*—The author details the history of a tabetic with decubitus of the penis, a rather rare location.

5. *Paralysis and Tabes.*—Mendel details the histories of three interesting cases of syphilitic infection which were followed by tabes and general paralysis.

(Vol. 24, No. 2, January 16, 1905.)

1. Clinical Examination Concerning Muscle Tonus. R. Link.
2. Concerning Some of the Nerve Elements in the Cerebellum of Different Vertebrates. M. J. Gurewitsch.

1. *Muscle Tonus.*—In a clinical examination of cases the author found that in those cases in which there were either nutritive changes or contractures due to an organic basis the tone was absent, while in voluntary contractures in hysteria and tetany the tone was present. (These results are relative.)

2. *Nerve Structure of the Cerebellum in Vertebrates.*—The Purkinje cells have more large branching dendrites and granules than the other cortical elements. The higher the development of the animal the more the branching of these cells. The Golgi and the basket cells are more numerous in the early development, and regress with the growth of the animal, and may have some relation to its early growth. The higher the function the more the development of the cell.

(Vol. 24, No. 3, February 1, 1905.)

1. The Pathogenesis of Choked Disc. A. Saenger.
2. The Relation of Tertiary Syphilis to Tabes Dorsalis and Progressive Paralysis. C. Hudovernig and J. Guszman.

1. *Choked Disc.*—Saenger reviews the various theories regarding the pathogenesis of choked disc, and combats the theory that the papillitis is due to a toxin generated by the tumor. He emphatically believes that intracranial pressure is the cause, and advises cranial relief by operation.

2. *Tertiary Syphilis and Tabes.*—The authors examined a large series of cases to determine the relation of the tertiary syphilitic stage to tabes and general paralysis, and concluded that in those cases in which syphilitic infection appeared within three years the nervous system was not diseased in 44 per cent. of the cases, and in 46 per cent. tabes or paresis was present. The relation in these cases of etiology to disease is evident.

(Vol. 24, No. 4, February 15, 1905.)

1. Hypophysis, Epiphysis and Peripheral Nerves in a Case of Cretinism. Bayon.
2. The Etiology of Nerve Degeneration. The Results of Transplantation Experiments. L. Merzbacher.
3. Is there a Pathological Plagiarism? O. Juliusburger.

1. *Cretinism.*—Bayou gives the result of an examination of the hypophysis, epiphysis and the peripheral nerves in the case of the cretin Ferdinand Stock. He made a large number of control preparations of the

pituitary body and the epiphysis, the latter being unsatisfactory. The hypophysis contains two parts, the anterior or glandular and the posterior division. The anterior part consists of nerve cells of three kinds, a lot of colloid substance which appears like vacriolated muscle plasm and intercellular substance. In the posterior part unmyelinated nerve fibers are found. In the cretin the nerve cells and the colloid substance were increased. The examination of the epiphysis was unsatisfactory. The peripheral nerves were not much diseased.

2. *Nerve Degeneration.*—The author, to determine the method of degeneration of nerves, transplanted nerves either to other parts of the same animal or to an animal of the same species, or to an entirely different kind of animal. The degeneration may be either a simple regressive process *in toto* or a necrobiotic process. His results were in auto-transplantation of a simple regressive kind, and in hetero-transplantation of a necrobiotic kind.

(Vol. 24, No. 5, March 1, 1905.)

1. The Question of Lumbo-Femoral Reflexes. W. v. Bechterew.
2. Concerning the Arciform Nucleus in the Medulla Oblongata and the Accessory Olives. G. Volpi-Ghirardini.
3. The Question of the So-called Choked Disc. A. Adamkiewicz.
4. The Meaning and the Influence of a Non-Salt Diet, and the Treatment of Epilepsy. L. J. J. Muskens.

1. *Lumbo-femoral Reflex.*—Discussion of reflexes.

2. *Medulla Oblongata.*—The author comes to the following conclusions: 1. The arcuate nucleus of the medulla oblongata is situated in the pyramidal region, sometimes near the sensory root of the fifth nerve. Kölliker's name of the "inverted pyramid nucleus" is, therefore, unnecessary. 2. Sometimes in the lateral tracts of the medulla oblongata are found nuclei which are not to be confounded with nuclei laterales. Because of their relation to the external arcuate fibers they belong to the nuclei arciformi. 3. By Nisal preparation they are composed of light-blue ground substance. 4. The ununiform size of these nuclei is explained by the fact that they are found exclusively in the genus homo. 5. Not always do these nuclei correspond to those of the pons. Whether their formation is analogous is a question. 6. The anomalous formation of the nucleus arciformis is analogous to other anomalies.

3. *Choked Disc.*—Responsive article.

4. *Epilepsy.*—The relation of salt diet to disease, and especially to epilepsy, is discussed.

(Vol. 24, No. 6, March 16, 1905.)

1. A Contribution to the Knowledge of Dissociation of Temperature and Pain Sensations in Injuries and Disease of the Spinal Cord. Preliminary Contribution. J. Piltz.
2. Neurological Examinations in Bicycle Riders. S. Auerbach.
3. A Contribution to the Study of the Transmission of Acquired Habits. G. Lomer.
4. Choreic Diplegia with Isolated Symmetrical Muscle Paralysis of an Atrophic, Flaccid Character. S. Klempner.

1. *Dissociation of Sensation.*—Piltz reviews the different theories regarding the course of the fibers for touch, pain and temperature in the spinal cord, and as a result of his own experience and of cases in the literature, believes that there is a distinct tract for pain and temperature, probably in Gowers' bundle. Dissociation of sensation of the syringomyelia type may be due to cortical, spinal cord and peripheral nerve lesions. In spinal cord lesions involving the posterior horns the upper limit of thermoanalgesia will be directly below the seat of the lesion on the same side. If the lesion involves Gowers' bundle, or if its fibers in the gray matter of the same side, the upper

limit of thesmoanalgesia will be about 5 to 6 spinous processes below the seat of the lesion or injury. This is important, as it indicates the limited extent of the lesion.

3. *Bicycle Riders.*—The author examined bicycle riders after exhaustion and found such examples of exhaustion as would probably be found in any series of men who were physically exhausted.

4. *Diplegia.*—The author records two rare cases of choreic diplegia. In one there was a bilateral atrophy of the rhomboidei, and in the other of the perineal group of muscles. No explanation is given.

(Vol. 24, No. 7, April 1, 1905.)

1. Electro-motor Qualities of the Finger. SOMMER.
2. Concerning the Arcuate Fibers of the Medulla Oblongata. L. JACOBSOHN.

1. *Electro-motor Qualities of the Finger.*—The author made some interesting physiological experiments concerning the electro-motor influences upon the body.

2. *Medulla Oblongata.*—Continued article.

(Vol. 24, No. 8, April 16, 1905.)

1. The Examination of the Light Reaction of the Pupil. O. VERAGUTH.
2. Some New Bone Reflexes in the Lower Extremities in Healthy and Diseased Individuals. J. VALOBRA and M. BERTOLOTTI.
3. Concerning the Arcuate Fibers of the Medulla Oblongata. L. JACOBSOHN.

1. *Pupillary Reflexes.*—The author devised a rather clever instrument which has as its purpose the projecting of a certain amount of light upon the retina. The light reflexes can be readily and comfortably studied.

2. *Bone Reflexes.*—The authors describe some new bone reflexes.

3. *Arcuate Fibers.*—The author considers rather extensively the arcuate fibers of the medulla oblongata. WEISENBURG (Philadelphia).

Allgemeine Zeitschrift fuer Psychiatrie

(Vol. 62, 1904, No. 3.)

1. Contribution to the Study of Katatonia. A. SCHOTT.
2. Simulation of Mental Diseases. H. BISCHOFF.
3. The Clinical Estimation of Pathological Roving. C. v. LEUPOLDT.
4. Late Recovery from Psychoses. JULIUS SIGEL.
5. Periodic Mania. FRIEDRICH GEIST.
6. Unusual Hypermnesia for Calendar Dates in a Low-Grade Imbecile. J. VAN DER KOLK and G. J. B. A. JANSENS.
7. The Mental Condition of Deaf Mutes. HERMANN KORNFELD.
8. Is the Abandonment of Alcohol as a Beverage in the Asylum Desirable? H. DEITZ.
9. Investigations on Dementia Precox, with a Suggestion as to Its Cure. GEORGE LOMER.

1. *Katatonia.*—The author relates the histories of two patients, males, aged respectively 29 and 35 years, in which at the start a diagnosis of neurasthenia was made, no definite mental impairment having been observed. After a persistence of neurasthenic-hypochondriac symptoms for two years in the one case and three years in the other, the advent of hallucinations and illusions, of ideas of persecution and unseen influence of negativism and stereotypy, with progressive dementia, stamped them as examples of katatonia. These cases emphasize the necessity for great care in the prognosis of neurasthenic and hypochondriac conditions, and for preventing our conception of neurasthenia from extending over too great a field. Considering the question of diagnosis, the author makes the following suggestions: Hypochondriac ideas in dementia precox are marked

especially by an absurdity and monstrosity, to which the affective condition does not correspond in depth or persistence. In the simple dementing forms these ideas are, as a rule, not projected into the outer world, though in dementia paranoides this takes place to a limited extent. Attempts at explaining them are from the start senseless and wanting in logic and stability. The emotional condition can temporarily reach a high grade of intensity, but speedily falls again to a level. Later absurd and ill-defined persecutory delusions which influence the psyche of the patient occur, and ideas of grandeur of equal monstrosity are not wanting. In the katatonic form, the delusional ideas are more persistent, and excite strong emotional outbreaks. A long-continued negativism with reaction by outbreaks of rage to the stimuli of the outer world is often observed, while beneath this condition, dementia makes rapid progress. Stiffness and mannerism in gesture and speech in a neurasthenic should be considered as suspicious from a diagnostic and prognostic point of view.

2. *Simulation of Mental Diseases.*—The author reviews the chief points which are to be considered in the determination of whether apparent mental disturbance is real or simulated, especially in persons accused of crimes, giving as examples four illustrative cases. A person may be not entirely normal mentally and still may simulate definite mental disease; in fact, simulation he thinks most common among the defective and criminal classes, many of whom, having been confined in institutions or associated in their own families with insane persons, are familiar with the symptoms of insanity. The chief means of deciding in any case are by the history, and by careful and constant watching while under detention. The manifestations of a definite form of mental disease can rarely be reproduced and kept up for any length of time without break or complication by some antagonistic symptoms. To the four cases mentioned above the author then adds an account of a habitual thief and swindler who was undoubtedly to some extent defective, and had had an injury to the head, and syphilis. During one of his terms in prison he acted in such a way as to raise doubts as to his sanity, and was committed to an asylum, where the diagnosis of beginning general paresis was made. Escaping after a fifteen months' sojourn, he plied his trade as usual and showed no serious mental defect. Coming under the author's care four years later, after a fresh crime, he appeared to have a certain degree of dementia, with loss of pupillary light reflex, diminished knee-jerk, staggering gait and tremor of the hands, while characteristic speech disturbance was absent. Careful investigation showed, however, that the mental symptoms had only appeared, and with some suddenness, since his arrest, and that when thinking himself unobserved his bearing was quite natural. Convinced that there was no dementia, the author considered the eye symptoms, diminished reflexes, etc., as due to the previous syphilis and pronounced the man mentally responsible.

3. *Pathological Roving.*—Considering the symptom of sudden giving up of the usual life and wandering off in a more or less aimless manner, presumably on account of some morbid impulse, which has been specially studied by Heilbronner and Schultze, the author combats the idea that all, or even a majority, of these cases are due to epilepsy, and shows that memory for occurrences during the period of wandering is not necessarily lost, but may be nearly or quite perfect. He gives the histories of three cases—from the Giessen Clinic—in which this pathological wandering was a feature. The first of these, after analysis, he decides to be an instance of defective personality developing upon a degenerative basis, and marked by excessive psychogenic reaction to moderate irritants, as slight annoyances, alcoholic indulgence, etc. In the second case the trouble seemed due to dementia precox with marked hallucinations. The third case did not show a definite clinical picture, but apparently was that of

a man of somewhat defective organization in whom the wandering was directly traceable to a period of alcoholic and other excesses.

4. *Late Recovery from Psychoses.*—Cases of late recovery from psychoses come occasionally into the experience of every alienist. The author takes up the subject in an attempt to find if possible upon what criteria as to form of mental disease or clinical manifestations we can base a favorable prognosis. What constitutes recovery? In Kreuser's opinion recovery is to be predicted when there are no more external symptoms, realization of past illness is present, and there is no defect, or only so slight a one that the patient can take up his usual life and calling as before his illness. As late recovery, he considers that occurring after three years' persistence of the psychosis. Opinions as to this point differ, however, considerably. Kraepelin thinks that while a few cases of late recovery occur in manic depressive insanity, the majority of such recoveries are in katatonia and are really partial only, some defect remaining. The latter opinion the author regards as too pessimistic. As to the form of psychosis in which late recovery takes place, study of the literature makes it appear that it is not limited to any one class of cases. Considering cause, cases with little or no insane heredity have in the main a better prognosis. Age and sex play a rôle to the extent that most of the cases of late recovery occur about the time of the climacteric. That acute diseases, trauma and change of surroundings seem sometimes to favor late as well as early recoveries is well known, but their action cannot be explained. Slowly occurring recoveries are more promising than sudden ones as regards permanency. Histories of three cases of late recovery are appended. The first, recovery from mania after eight years, female. The second, hallucinatory periodic disturbance, recovery after four years, female. The third, hysterical insanity, recovery after nine years, female. The author concludes that while late recoveries are not so rare, we cannot so far single out any special symptoms or stigmata which will enable us to predict probable recovery in any given case.

5. *Periodic Mania.*—Report of a case of periodic mania in which six attacks had occurred, between them intervals of perfect lucidity extending from six months to nine and a half years. The later attacks showed a tendency to last longer, the sixth thirty-four months, followed by correspondingly lengthened lucid intervals, the last nine and a half years. The patient at the time of the report had been for nearly two years in a seventh attack, during which, however, he had had short intervals of lucidity lasting from four to fourteen days. No nervous or insane heredity could be made out, nor was there any evidence of alcoholic abuse, but during boyhood the patient had had several attacks of illness, one a febrile condition, lasting some time. The author thinks that the mental diseases may have been due to some brain involvement on one of these occasions.

6. *Hypermnesia for Calendar Dates in a Low-Grade Imbecile.*—Cases of unusual ability in some narrow channel, depending mainly upon extraordinary memory within a limited circle, in persons otherwise very deficient are exemplified from time to time in the various natural musicians, "reckon artists," etc. The "lightning calculator," Inaudi, has been studied carefully by Binet, who found him incapable of learning anything outside of his specialty, and other cases have been described by Wizel, Falret, Forbes-Winslow, Moebius and others. The authors add an account of a case of extraordinary memory for calendar dates relating to birthdays, fetes, etc., extending over the two previous years, in an imbecile of such low grade that no satisfactory psychological examination of him could be made, the patient being unable to comprehend what was wanted of him. This man worked in the mattress factory of the asylum, but could never be taught more than to fetch and carry. He had an extraordinary fondness for calendars, of which he collected all possible, making piles of leaves which he aparently studied continually. He could talk, and

answered promptly questions relating to dates, but otherwise was incapable of learning anything.

7. *Mental Condition of Deaf Mutes.*—The author declares that the matter of mental responsibility of deaf mutes is treated in text-books on legal medicine in "right stepmotherly" fashion. To illustrate the problems involved and the opinions prevailing, he describes the procedure in two cases of deafmutism, the first that of a woman accused of child-stealing, drunkenness and general immorality, the second that of a woman who exposed her new-born illegitimate child in a cemetery. The first case, after consideration of all the facts, was prouounced responsible in a legal sense, but as she was shown to be of defective mentally and of low moral instincts, her commitment to an asylum was recommended. She had been at a deaf mute school for six years, had learned to read and write and to sew, and presumably should have imbibed some moral principles. The second woman had never been to school or had any instruction, and could neither read nor write. Considering all the facts in the case, the medical examiner decided that while she probably had some idea of right and wrong, she was incapable of appreciating her act in all its enormity and should be committed to an asylum. The author insists that a distinction should be made between those who are deaf and dumb from birth and those who become deaf in early childhood, since there is strong presumption that in the first class there is defective brain development. Also there is a difference between instructed and uninstructed deaf mutes, since in these unfortunates the usual avenues for the imbibition of moral and ethical principles are closed and special treatment is necessary to make them orderly and helpful members of the community.

8. *Alcohol as a Beverage in the Asylum.*—The rôle of alcohol in producing and keeping up mental diseases, both in users and their descendants, has apparently little need to be emphasized, but in Germany the custom of allowing certain of the patients a modicum of wine or beer as a special indulgence has long prevailed. To this practice the author has, as a result of own experience become opposed. He calls attention to the difficulty of enforcing total and habitual abstinence in the large class in whom it is necessary in an institution where it is allowed to others. Among those necessarily to be kept abstinent he classes alcoholics, the alcoholic insane, epileptics insane and otherwise, arteriosclerotics, paretics and idiots. In the Hessian Grand Ducal Asylum at Goddelau he found this class to make up 40 per cent. of the population, 27 per cent. consisted of those too demented to miss their stimulant, leaving only 33 per cent. in whom the cutting off of beer seemed likely to create dissatisfaction. Even among the latter class, after several months of teetotal regime, only a small number, not over 5 per cent., continued to grumble about it. The author thinks the results fully warrant giving up all alcohol as a beverage in the asylum, not to speak of the saving of money, which sum he suggests, can well be applied to the purchase of other extras. He would prohibit alcoholic beverages to both patients and personnel.

9. *Dementia Precox.*—The author thinks that it is evident that dementia precox is in some way connected with the sexual function, probably is due to an excessive or deranged internal secretion of the sexual glands. He gives the results of his study of 86 cases, all in females, which he thinks shows a close relation between the disease and the functions of menstruation, ovulation and reproduction. Basing his opinion upon these considerations and referring to the good results said to have been obtained by Goodell from oophorectomy in cases of menstrual psychoses as long ago as 1882, he suggests the removal of the ovaries in female precocious dements as soon as a positive diagnosis is made.

ALLEN (Trenton).

Miscellany

A CASE OF RAPID PARAPLEGIA. G. R. Hamilton (Liverpool Medico-Chirurgical Journal, No. 46).

The author reports the case of a man, 41 years old, who had a trifling fall from his bicycle on August 27. That it was not a serious accident is plain from the fact that he walked twenty miles, later in the same day. He was as well as usual until the night of October 27, when he complained of having contracted a "chill." He travelled to London and, not feeling well, returned to Liverpool on October 30, on the evening of which day he was seen by the author. He seemed to have some intestinal catarrh and a cold. Some gray powder was ordered. The next evening his temperature was 101 degrees, and the same the following day. He was kept in bed. The following two days showed improvement, with normal temperature, clear tongue and a desire to go to work. At four on the same afternoon, while talking cheerfully he found he had suddenly lost the use of his legs. Before he could get to bed he was completely paralyzed from the waist downward. The author saw him that afternoon and found complete motor paralysis and loss of sensation from the level of the ninth dorsal nerve downward. The line of sensory appreciation slightly above the umbilicus was distinctly marked. There was no line of hyperesthesia. Plantar, patellar and cremasteric reflexes were absent. He had an evening temperature of 101 degrees, with severe headache, and pain in back. The abdomen was distended from intestinal paralysis and there was retention of urine. On the morning of Nov. 5 he felt better, and the urine which was drawn off contained no sugar nor albumin. On the afternoon of that day, however, the loss of sensation had extended upward to the level of the spine of the scapula. At midnight he suddenly became unconscious and the respirations had increased. In the morning the breathing was entirely diaphragmatic. His face was turned to the left, and there was varying deviation of the eyes to the left. He nearly choked on attempting to swallow a little water. On Nov. 7 both arms were paralyzed. Right facial paralysis was present, but was overcome, together with the deviation of the eyes by the fresh administration of oxygen. He died on Nov. 9. The diagnosis from the first was that of hemorrhage. The lesion was probably located in the lumbo-sacral region, although difficult to locate definitely. The author thinks the accident had nothing to do with it. The high temperature pointed to a septic origin, but the feverishness entirely disappeared before the paraplegia occurred. Post-mortem the lesion was discovered to be intramedullary hemorrhage, most marked at the level of the ninth dorsal nerve. At this point the cord was almost entirely destroyed, and the hemorrhage extended down to the level of the third lumbar vertebra and upward to the mid-cervical region. JELLIFFE.

THE GROSS APPEARANCES OF THE TISSUES OF THE IRIS IN EPILEPSY. C. A. Oliver and J. C. Knipe (The Ophthalmoscope, May, 1905).

The material for these studies was obtained from the nervous wards of the Philadelphia Hospital. The work was done mostly during the writers' last three months of service, in the ophthalmic wards of the institution (January, February and March of last year, 1904).

Every case was submitted to a critical ophthalmic examination, in order that all local ocular disease might be excluded. Nearly 60 subjects, males as a rule, and preferably native-born American adults, were used. Each eye was examined in every relevant detail. Notes of all the findings were taken at the time, and later compiled as a whole without any regard to the character of the results. Diffuse daylight was concentrated upon the magnified tissues of the membrane in such a way that thorough study could be made of every exposed portion. As a result it was found:

First, that the pupils, like those of normal individuals, were oval or ovoid, with the angles of their long axes placed slightly downward and outward.

Second, the pupils, as a rule, were unequal in size, that of the left eye being generally larger.

Third, the pupillary rims of the pupillary zones presented unusual degrees of uveal pigment fringing.

Fourth, the muscle areas of the pupillary zones were, as a whole, rather deeply tinted, and the composite fibers appeared to be slightly thickened.

Fifth, the divisional minor circles were not distinctly outlined, their interlacings and crypts in most instances not being sharply and clearly defined.

Sixth, the radiary fibers of the ciliary zone were plainly marked and outlined, although the intervening minute depressions were blurred and indistinct in some places.

Seventh, the concentric contraction grooves in the ciliary zones were abruptly broken in places, with a lessening of the indentation depths and an undue broadening and elevation of the corresponding furrow ridges.

Eighth, the vascular spots and pigment aggregations ordinarily seen in the ciliary zone were probably increased in number and size.

Ninth, the peripheral pigment in the generally invisible ciliary rim of the ciliary zone was sufficiently broadened in some situations in some of the cases that it could be seen under oblique illumination.

J. E. Clark (New York).

INSANITY AT PUBERTY. C. W. Burr, Philadelphia (Journal A. M. A., July 1).

Dr. Burr states that he considers everybody somewhat unbalanced at the age of puberty, but that only the weaklings develop mental disease. What is probably the most common type of this period is seldom seen in asylums, and has been but little studied. It consists in a mild general impairment—dementia is too severe a word, but it seriously affects the future life of the individual. In another type there is gross moral aberration in addition to the mental deficiency (which may be slight). Many of the criminal class are of this type. Another form which begins at puberty or early adolescence is the so-called original paranoia. In all these types the prodromal symptoms appear at puberty, the disease reaching its height in adult life. Acute insanity also appears at puberty, and though rare, is sufficiently frequent to be of interest. It may follow acute infectious disease or mental shock, and illustrative cases are reported by the author. Kraepelin's dementia precox is mentioned as a rather comprehensive term, but one that must be convenient in grouping certain classes of cases. A case of apparent stuporous insanity is described in this connection. Juvenile paresis is recognized as occurring in hereditary syphilis. The influence of other organic disease, Burr claims, has been studied but little. Acute insanity is not uncommon in imbeciles, and usually occurs about the adolescent period. Hysteria may give trouble in its diagnosis from dementia precox, and here the study of the visual field is of value. Hysteria is not followed by dementia. Its chief significance is as a sign of trouble in later life. The treatment of insanity at puberty is largely preventive, such as proper education, avoidance of mental strain, teaching of self-control and clean morals. With more wisdom of parents insanity would decrease

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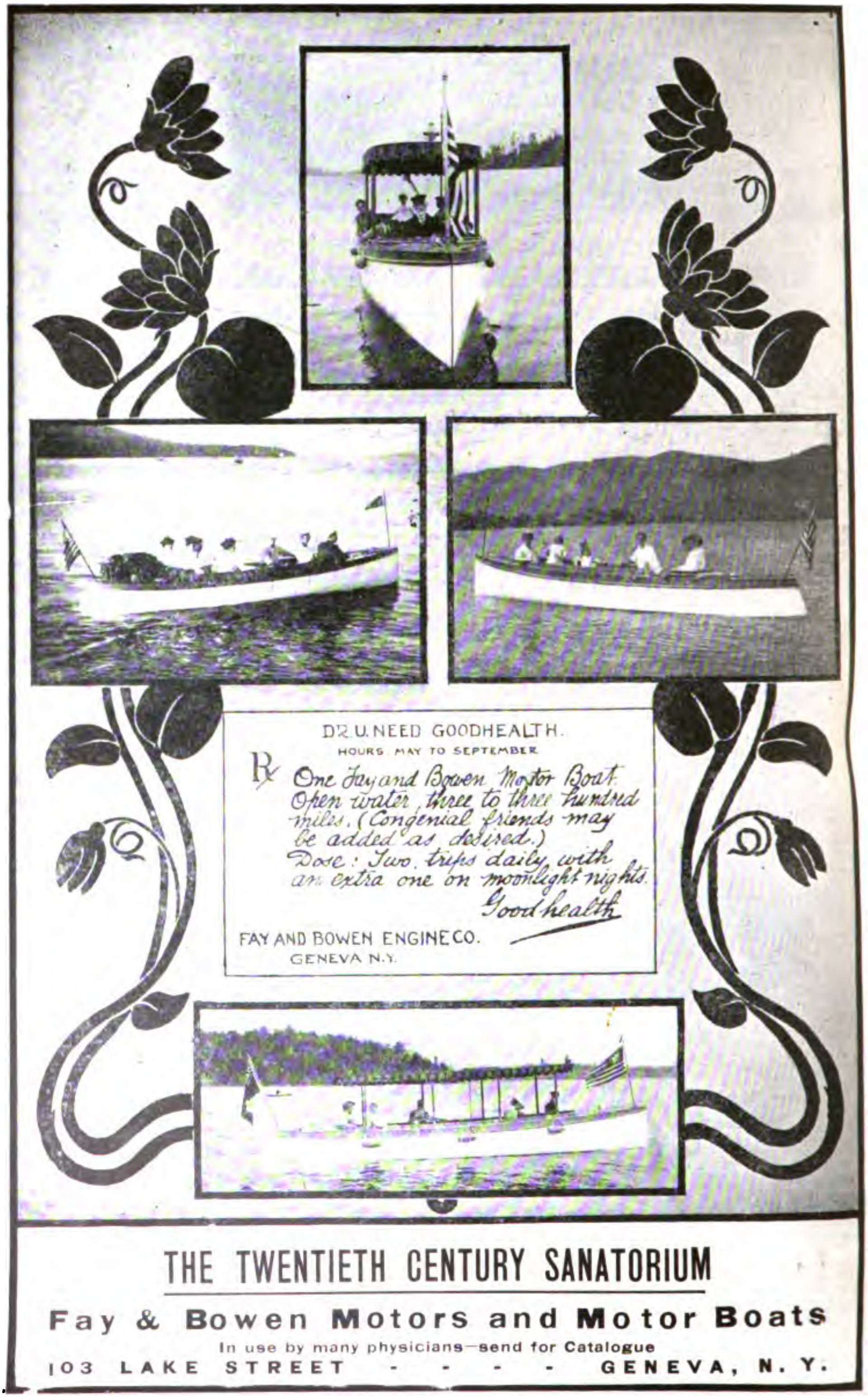

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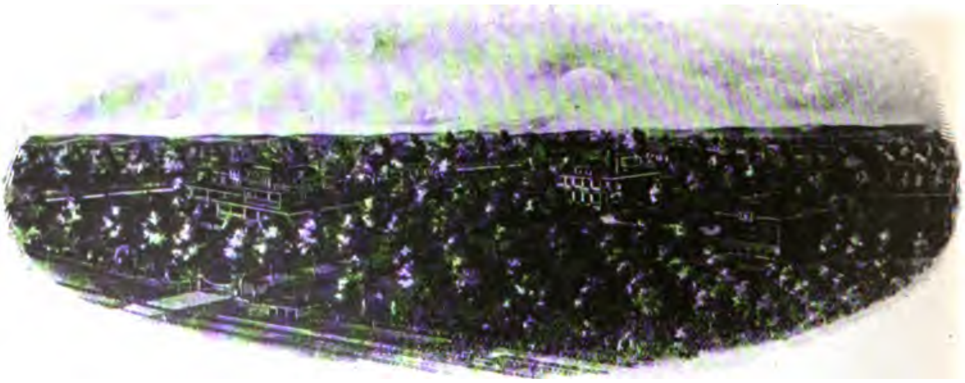

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THE
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Original Articles

THE DIET IN EPILEPSY.

BY A. J. ROSANOFF, M.D.,

OF KING'S PARK STATE HOSPITAL, NEW YORK.

It is undoubtedly a well-known fact that diet has an important influence on the manifestations of epilepsy, and medical literature contains numerous statements concerning the effect of one or the other kind of food upon the frequency and severity of the seizures. Most authorities agree that meat in large quantities is harmful to epileptics. Consequently, dietaries prescribed for such patients include, as a rule, a relatively scant allowance of meat, and that given usually in one of the daily meals only.

The effect of varying, not the kind of food but its proximate composition, has not been subjected to systematic study directly. Some interesting data may, however, be found in a well-known communication by Merson.[1] To test a hypothesis which had been advanced by Hughlings Jackson, according to which a palliative for epilepsy might be found in the substitution of phosphorus for some of the nitrogen in the chemical make-up of cortical tissue, Merson kept a group of twelve epileptic patients for four weeks on a "farinaceous diet," poor in nitrogenous principles, but containing a normal amount of fats and a large amount of carbohydrates (proteids, 74 grams; fats, 92 grams; carbohydrates, 312 grams, daily); these patients were receiving, in addition, ten drops of phosphorated oil daily. A similar group of patients was kept on a "nitrogenous diet," containing an excess of nitrogenous material, but insufficient in fats and carbohydrates (proteids, 202 grams; fats, 45 grams; carbohydrates, 215 grams, daily). The effect of the farinaceous diet was an unmistakable decrease, that of the nitrogenous diet—a marked increase, in the frequency of convulsions. The same effect was obtained when, in a second experiment, the first group of patients received the nitrogenous, the second group the farinaceous diet.

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[1] J. Merson. The West Riding Lunatic Asylum Medical Report, 1875.

Ample experience since has shown that phosphorus is entirely worthless in the treatment of epilepsy. Merson's results, therefore indicate that either the reduction of nitrogenous matter or the reduction of meat has a beneficial influence in epilepsy.

Turning from the empirical results of dietetic investigations to the recent chemical-pathological studies of epilepsy, there is good ground for the assumption that the various manifestations of this disease are probably dependent upon a disorder of nitrogenous metabolism, and upon nothing else. This disorder is supposed to consist in the formation of a poisonous substance, which is either not formed at all in the normal state or exists only in harmless quantities, and which in epilepsy accumulates in the body more or less rapidly, causes vertigo, convulsions and psychic equivalents, and during the seizures is converted into urea—the final product of proteid metabolism. In other words, in epileptics, at a certain stage in the process of conversion of proteid material into urea there is a hitch, and abnormal, often violent, activity of the nervous and muscular tissues becomes necessary for the completion of this process of conversion.

That a substance capable of producing convulsions and other phenomena of epilepsy actually exists in the blood of patients in the status epilepticus has been proved by the researches of Krainsky who, by injecting blood obtained from patients in that condition into guinea pigs, produced convulsions and paraplegia. The same observer also found in the blood of patients in status epilepticus large quantities of ammonium carbamate; and he has shown that by injecting this substance into rabbits epileptic seizures can be produced.[2]

Ammonium carbamate is said to be present in the blood normally in very small quantities and is supposed to be a precursor of urea. Its conversion into urea occurs chiefly in the liver, and Krainsky finds further corroboration of his theory, according to which the phenomena of epilepsy are due to the periodical accumulation of ammonium carbamate in the circulation, in the experiments of Hahn, Massen, Nencki and Pawlow. These observers have shown that when in animals an Eck's fistula is established (an artificial communication between the portal vein and the inferior vena cava), the portal circulation is thrown out of action and the ammonium salts formed in the course of proteid catabolism, being no longer completely converted into urea, accumulate in the circulation, presumably in the form of the carbamate, and give rise to convulsions, ataxia, somnolence and coma before the animal dies.[8]

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[2] Binswanger, "Die Epilepsie," p. 238, *et seq.*

In the light of these experimental facts, it seemed to me safe to interpret Merson's observations in the sense that it is not the kind of food, not meat or farinaceous material as such, but the absolute quantity of proteid matter, irrespective of its origin, that constitutes the dietetic factor of influence on the frequency and severity of epileptic manifestations. In view, however, of the great practical importance of the question, I decided to subject this interpretation to the test of direct experiment.

Eleven patients suffering from "genuine" epilepsy of old standing, but presenting no complications, were kept on five different diets consecutively, the kind and content of the food being systematically varied. For the purpose of eliminating the influence of exercise on metabolism, the patients were kept in bed during the entire duration of the experiments. For a similar reason they received absolutely no medication. A careful record was kept of the weight and mental condition of the patients, as well as of the number of convulsions. Since the frequency of the seizures in any case of epilepsy is apt to vary without any apparent cause, I thought it wise to compare the total weekly average number of convulsions under each diet, and not, for instance, the number for each patient for each week. Following are the results:

EXPERIMENT I. REGULAR DIETARY OF THE HOSPITAL.

Breakfast: Oatmeal, wheat flakes, or farina with syrup or milk; bread, butter, coffee with milk and sugar.

Dinner: Beef, mutton, veal or pork; potatoes, vegetables, bread.

Supper: Pudding, cake, baked beans or macaroni; fruit, bread, butter, tea with milk and sugar.

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[8] Hahn, Massen, Nencki and Pawlow. "Die Eck'sche Fistel zwischen der unteren Hohlvene und der Pfortader und ihre Folgen für den Organismus," Archiv für experimentelle Pathologie und Pharmakologie, Vol. 32, p. 18.

This dietary is somewhat variable, and the quantity of food received by each patient is not accurately measured; there is, however, no important variation in the proportions of the different proximate principles. The amount of proteids is small, that of the carbodydrates large, and that of the fats moderate.

This experiment was carried on for ninety-one days (from Dec. 1, 1903, to Feb. 29, 1904). During that time the patients' mental condition presented no unusual feature; their weights remained nearly stationary, and the total average of convulsions per week was 14.3.

EXPERIMENT II. VEGETABLE DIETARY.

Breakfast: Oatmeal, wheat flakes, or hominy with syrup; bread, butter, coffee with sugar, but without milk.

Dinner: Beans or peas, rice, sago or tapioca, potatoes, vegetables, bread, butter.

Supper: rice, cornmeal or bread pudding, fruit, bread, butter, syrup, tea with sugar, but without milk.

This dietary differs from the regular hospital dietary only in the substitution of vegetable proteids for those of animal origin, the quantities of the proximate principles being practically the same in both. This experiment was carried on for forty-six days (from May 1, 1904, to July 15, 1904). During that time no change from the usual mental condition of the patients was observed. Seven patients gained in weight from three to eight pounds; one patient lost three pounds, and the weight of the remaining three patients remained constant. The total average of convulsions per week was 14.3.

EXPERIMENT III. DIETARY WITH INSUFFICIENT QUANTITY OF PROTEIDS.

Breakfast: Rice, 3 oz. (weighed before cooking); milk, 8 oz.; sugar, 1 oz.; coffee.

Dinner: Rice, 3 oz.; milk, 8 oz.; tea.

Daily quantities of proximate principles received by each patient: Proteids, 62 grams; carbohydrates, 336 grams; fats, 41 grams.

This experiment was carried on for forty-seven days (from July 16, 1904, to Aug. 31, 1904). During that time the patients were restless and irritable; their weights remained stationary; the total average of convulsions per week was 17.6.

EXPERIMENT IV. DIETARY WITH EXCESSIVE AMOUNT OF PROTEIDS.

At each meal, 2 eggs; milk, 24 oz.; bread, 3 oz.; sugar, 1 oz.; tea or coffee.

Daily quantities of proximate principles received by each patient: Proteids, 148 grams; carbohydrates, 305 grams; fats, 116 grams.

This experiment was carried on for thirty days (from Sept. 1, 1904, to Sept. 30, 1904). All the patients gained in weight from four to twelve pounds. Mentally they were restless and irritable. The total average of convulsions per week was 18.2.

During October, 1904, the regular diet was resumed. The total weekly average of convulsions then fell to 14.

EXPERIMENT V. DIET WITH LARGE EXCESS OF PROTEIDS AND VERY DEFICIENT IN CARBOHYDRATES (THE DIABETIC DIETARY OF THE HOSPITAL).

Breakfast: Eggs, bread, butter, coffee.

Dinner: Meat, cabbage, lettuce or celery, bread, butter, coffee.

Supper: Eggs, stew or cheese, bread, butter, tea.

No milk was used. Saccharin was used with the tea and coffee. Where bread is called for on the dietary bran bread or gluten bread was used.

This experiment was carried on for twenty-one days (from Feb. 13, 1905, to March 5, 1905). Most of the patients' weights were diminished from two to five pounds. Mentally, they became restless, irritable, noisy, destructive to their clothing, and assaulting. The total weekly average of convulsions was 23. The increase in the number of convulsions persisted for about two weeks after the regular diet had been resumed.

To sum up the results: 1. Experiments I and II prove that the effect of a mixed diet in epilepsy differs in no way from that of a vegetable diet containing the same quantities of proximate principles. Consequently any lingering belief that animal food has any effect as such is to be definitely discarded.[4] 2. Experiments III and IV prove that the quantity of proteids in the diet has a decided influence on the manifestations of epilepsy; if it is either above or below the indispensable minimum the severity of the disease is increased. 3. Experiment V shows that if the diet of an epileptic is made to contain a large excess of proteid and practically no carbohydrates, so that the organism is compelled to use proteid material in place of carbohydrate material, the number of convulsions increases enormously and there is a general aggravation in the physical and mental condition of the patient.

It seems, then, that *just as the organism of the diabetic is unable to properly utilize carbohydrates, so the organism of the epileptic cannot take care of proteid material as it is taken care*

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[4] In this connection it may be interesting to note the fact that epilepsy is common in the herbivora.

of by the normal organism. The epileptic is, however, the worse off of the two, for while the diabetic can without serious harm leave out carbohydrates from his diet, substituting for them fats and proteids, the epileptic can only partly substitute proteids by the other proximate principles, being compelled to take a certain minimum amount to sustain life.

Possibly the analogy between diabetes and epilepsy can be carried still further. The cases upon which my experiments were made are of old standing, with the "epileptic habit" deeply rooted in the organism, with mental complications, and possibly with secondary organic changes in the nervous system; they may be compared to "severe" cases of diabetes in which even the most rigid diabetic restrictions fail to cause a total disappearance of the manifestations of the disease. On the other hand, there are mild cases of epilepsy, which are not of long duration, in which the seizures are infrequent and which are not complicated with mental disorders; these may be compared to "mild" cases of diabetes in which it is unnecessary to exclude completely carbohydrate substances from the diet, a mere reduction in this quantity being sufficient to remove all the evidences of the disease. These mild cases of epilepsy may be reasonably expected to recover completely under appropriate dietetic treatment in accordance with the principle laid down above.

The therapeutic indication is clear. Carbohydrates and fats are to a certain extent capable of replacing the proteids in the diet.[5] The epileptic patient, then, should receive the largest amount of carbohydrates and fats that he can assimilate without inconvenience, and the smallest amount of proteids which is compatible with the preservation of the nitrogenous equilibrium; that is to say the amount of nitrogen ingested with the food must not be allowed to fall below the amount excreted, for then the thing is overdone, a condition of proteid starvation is established, the general health of the patient suffers, and his disease becomes aggravated.

---

[5] Herter, "Lectures on Chemical Pathology," p. 150.

EPIDEMIC MULTIPLE NEURITIS OF OBSCURE ORIGIN.*

BY M. A. BLISS, M.D.,
OF ST. LOUIS.

During September, 1904, at the request of the Suprintendent of the State Insane Asylum No. 4, located at Farmington, I went to the institution to examine twenty-four cases of what proved to be multiple neuritis.

The institution is a new one, built on the cottage or detached building plan, is well located on high ground, with good drainage, and supplied with an abundance of water from two wells 400 feet deep. Mosquitoes are not numerous, and malaria is very rare.

I found the patients well-fed and clean, and their general care intelligently provided for. Most of them had recently been brought from an older institution located at Fulton.

The total number of patients located in the asylum was 250. Of these 50 were epileptic. There were 23 men and 1 woman with multiple neuritis; 14 of them were epileptic.

Dr. Keith, the Superintendent, described the beginning of the cases as follows: The patient would be noticed to drag a little in gait, and later would show distress; if mentally competent enough, would complain of pain and fatigue in the legs and feet.

On examination, tenderness on pressure, flabbiness of the muscles of the legs, and in some instances atrophy of the muscles would be made out; in others edema, followed later by atrophy.

No gastrointestinal disturbance and no lesions of the heart. A rapid action of the heart, not accompanied by murmur or by rise of temperature, was nearly uniform.

In a week from the appearance of the first symptoms the patients were confined to bed or to a chair. Foot-drop, increased edema, greater tenderness in the feet and legs, and in the woman wrist-drop, occurred.

None seemed very sick. After a month or six weeks the process would reach a stationary stage, lasting another month, when recovery would begin.

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*Abstracted from a paper read before the St. Louis Neurological Society, May 29, 1905.

I had the opportunity to see them only once, and examined the entire number on the same day. A battery was, unfortunately, not available, so no electrical tests could be made. Neither could any blood examinations be made, because of lack of equipment.

In practically all the knee-jerks were totally abolished, even on reinforcement, the Achilles jerk corresponding to the knee-jerk.

The plantar reflex was abolished or delayed. The elbow-jerk and wrist reflex were affected only in those with arm involvement. Of these there were only two.

The pupils were not affected.

No sphincter relaxation, except in one very much affected patient, whose mentality was so lowered that examination was unsatisfactory.

Sensation: All evinced tenderness to pressure over the muscles most involved, especially the calves of the legs. The sciatics were not tender.

Pain sense was lowered or delayed in the severest cases, especially in those showing the greatest motor weakness, with foot-drop.

In no case was pain sense absent.

Tactile and temperature tests were very unsatisfactory, owing to the mental deficiency of those most severely affected, but in a general way seemed to correspond to pain.

In nearly all there was edema of the feet and ankles, and in a few, of the upper extremities, but none had edema of the trunk. A clammy sweat accompanied the edema. In some edema occurred subsequent to the atrophy.

Some of the cases were reaching the stationary stage when I saw them, and Dr. Keith told me the atrophy was greatest in those in whom the edema had been most pronounced.

In those able to walk the gait was in some instances a "shuffle" and in some a typical "high steppage."

The cases had been appearing for two months previous to my visit up to ten days before, none occurring after that. It required two months in all cases to reach the stationary stage, or that of slight betterment.

The patients were all pale, and judged by the appearance of the gums and conjunctiva, anemic, but as stated, no blood counts could be made nor the percentage of hemoglobin ascertained.

Of the 24 cases 7 have since died from intercurrent causes: pneumonia, nephritis and status epilepticus. I saw on May 24, 1905, all those still living and all had made either complete or fairly complete recovery except the woman, who still shows weakness of the extensors of the wrists and feet.

SUMMARY.

Slow onset, only lower extremities affected in most instances. Tenderness, easy fatigue, shuffling gait, early atrophy, sometimes preceded, sometimes followed by edema. Rapid heart action without murmurs; no gastrointestinal disturbance. Fairly complete recovery in eight months in those surviving intercurrent troubles, none dying during the advance of the neuritis.

I was unable to determine the etiology.

Lead could be excluded (no "lead line," obstinate constipation or colic; rare involvement of musculo-spiral or of any of the nerves of the upper extremities).

Arsenic could be excluded from consideration through lack of gastro-intestinal disturbance, and great intensity of pain. Arsenical cases I have studied have been slower to recover.

The large number of epileptics, 14 of 24, made me think there might be some explanation in that condition. I found they had been given a considerable amount of bromidia, but as none of the ingredients in the published formula of that preparation has been known to produce neuritis, I could look upon it only as a contributing cause. The cases ceased occurring just at the time a change was made in the medicinal regime, but it may be suggested that was merely a coincidence.

The known occurrence of multiple neuritis from drugs administered medicinally, as sulphonal and trianol, as well as arsenic, would lead us to think of the possibility of over "strenuosity," especially in epileptics, of medicinal administration.

Discussion of Bondurant's cases at Tuscaloosa, 1897.

Discussion of the Tewksbury Almshouse cases occurring this year.

In connection, the discussion, with a description, of the cases of beri beri occurring among the Filipino tribes exhibited at the Fair.

A STUDY OF DEMENTIA PRÆCOX.

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(*Continued from page 712*)

Katatonia—This type of dementia præcox originally conceived by Kahlbaum[83] in 1874 and by him recorded with monographic completeness as a clinical entity, Spannungsirresein or Katatonie, has at the hands of Kraepelin been deprived of some of its dignity and deposed to the rank of a symptom-complex, the katatonic symptom-complex. That even in this lesser rôle it has been grossly neglected in the English writings is best illustrated by the fact that only a meagre paragraph of some twenty lines ranged under the caption of stupor, is set down in Albutt's[84] System of Medicine. The author's justification rests with the universal feeling among English alienists against the acceptance of katatonia as a phenomenon of any importance. That Kraepelin's masterly dissertation on the clinical features of katatonia should have found such small favor with the English school is little short of amazing.

Irrespective of a consideration of the weak-mindedness, which sweeps inevitably towards its predestined goal of absolute dementia, this form of præcox demands for its adequate presentation, a thorough regard for many distinguishing motor phenomena and conspicuous acts of negativism, stereotypy and automatism. These manifold anomalous functional disturbances arising in either the initial stages of stupor and excitement, combine differently in different cases and may even show a marked variability in each individual case.

The writers, who have based their deductions on worthy material have failed to divulge a significant etiology. Hecker seemed to think that the physical and psychical upheaval incident to puberty exerted the most powerful influence. Illberg[85] remarks that of his cases 45 per cent. gave evidence of a bad heredity, and of these 30 per cent. showed the transmitted tendencies. This author, whose material included about twenty classic cases, states that the average onset was at about the age of twenty-four, the earliest having occurred at fifteen and the latest at thirty. Whereas many overly bright, promising youthful persons come under the ban of this affliction, the popular belief that precocious mental endowment is an essential prerequisite to the development of this form is not borne out by the facts. In Illberg's cases just 50 per cent. were talented beyond their years, and the others were not even possessed of average faculties. It was his experience that most of the patients were of the quiet, unresponsive, unobtrusive sort, in whose developmental years there had appeared data of some etiologic consequence; but of a direct relationship between cause and effect nothing convincing has been admitted. Fully one-half of Illberg's cases of katatonia occurred in students, merchants and clerks, and he agrees with others in the comment that "brain-workers" seem especially prone to this form of psychosis; 25 per cent. of his cases were recruited from various professional classes in the smaller towns and the other one-fourth were comprised of factory workers and general laborers. Many histories disclose the facts of great grief, unrequited love, excesses in "Baccho et Venere," and onanism in remote causal relation to the disease.

Conclusions as to the character of the onset in this disorder must be ventured only with great reserve, since neurasthenic symptoms and all manner of vague temperamental disturbances may precede by months what appears to be the first real mental storm. This gives to the onset a subacute complexion, which is the rule. Having touched upon delusional and illusional symptoms, the judgment, memory, thought-sequence and general conduct, in the earlier paragraphs of this paper, it might be well to forego needless repetition and discuss a few of the special katatonic symptoms.

In the beginning of the disease, there are but very few symptoms at variance with those described under hebephrenia —but in some cases, Kraepelin says in one-third at least, two mental states may without warning come over a patient, viz.: katatonic stupor or katatonic excitement, closely allied, although externally diverse. The stage of excitement is perhaps the more frequent of the two, but appears with no definite regularity or sequence. When the stages do not deliberately and plainly succeed one another, their elemental parts may oppose one another with such swiftness, that the patient is one moment swayed by excessive excitement and the next thrown into profound apathy. According to Kraepelin, the chief component factors in the establishment of katatonic stupor are automatism and negativism. One patient, whose history must for personal reasons not be related "in extenso," was in this stuporous state when seen for the first time. It was impossible to get an intelligible phrase out of him, nothing more than a low mumble; to engage him in conversation was out of the question—even efforts at monosyllabic talk resulted in failure. He took no notice of any form of address. Pricking him with a pin was without effect, and even energetic tunneling with a needle failed to induce a resistant attitude. During the entire examination, repeated fine twitchings were in evidence around the corners of the mouth.

Associated with the negativism already more or less specifically alluded to in the earlier parts of this paper, is muscular rigidity, which may attain a most extraordinary degree. It is this somewhat grotesquely and bizarre appearing muscle tension, which received distinction at the hands of Kahlbaum in the compound title Spannungs-irresein. The handshake of a katatonic patient is stiff, slow and automatic; there is no attempt made to grasp or hold the hand, but the whole extremity in semi-flexion is rigidly projected forward and held motionless until released, when it is withdrawn in the same inflexible manner. In the vast majority of cases, the muscular movements are deliberate and constrained, but by some patients they are executed with great suddenness and flashlike rapidity. In walking about, many keep stiff at the knee; others march on tip-toe with body inclined forward or backward in a most outlandish, ungainly fashion. The facial musculature participates conspicuously in the generalized tension and the features often appear mask-like, immobile, even to the absence of eye-blinking. The snout cramp (Schnauz Krampf) in which the patients pucker up their lips and protrude them rigdly, allowing a few short lightning-like twitchings to play through them, is so often seen in these cases as to be characteristic. (See Fig. 2, Case of G. B.) The trunk may incline at any angle and the extremities be projected wherever they are put, remaining in the most extraordinary and difficult poses for hours at a time.

In strong contrast to acts of muscular opposition as expressed in terms of negativism and muscular rigidity are the symptoms that indicate increased susceptibility to external impressions,—the cataleptic phenomena.

In the periods of katatonic excitement that alternate with those of stupor, patients persist in doing the most reckless, senseless, violent things; their movements are stereoptyped; their acts impulsive, destructive, even self-mutilating and during these times their general deportment is exceedingly bad. Kraepelin's assertion that these patients play with their excreta, drink their urine, wash themselves in it, eat feces and are guilty of other filthy practices is in no sense an exaggeration.

The confluent, syllable-stumbling type of speech-defect so characteristic of paresis does not occur in the katatonic form, but the tone used is unnatural, and of the sing-song, scanning order that reminds one forcibly of multiple sclerosis. Kraepelin notes that in certain cases where the ability to form whole sentences, however short, is lost, the infinitives alone are spoken, and this he calls "agrammatism." Verbigeration occurring as a symptom in this as well as other mental states, becomes evident in both the spoken and written language. (Neisser,[36] *Arch. f. Psych.*, Vol. 46, 170.)

The motor stereotypy which presents in the course of the period of excitement, occurs also in certain other maniacal, paranoid and demented states. Aside from the principal peculiarities of the katatonic type and the rapid onset and development of mental and moral insanity, there occur innumerable somatic symptoms that are, however, conceded as common to other forms of mental disease. I refer to the presence of emaciation, anemia, congestions, cyanosis, local edemas, abundant salivation, local or generalized erythematous blushings (of vasomotor origin), generalized hyperidrosis (without apparent cause), dermographia, anesthesia and analgesia. On the part of the motor apparatus, fibrillary twitchings, circumscribed tonic, clonic twitchings, epileptoid, apoplectoid and hemiplegic attacks are often noted. The widely dilated pupils react briskly to light. Polydipsia, polyphagia and polyuria are not infrequent. Cardiac and respiratory arhythmia are observed, as are also menstrual irregularities, causeless protracted diarrheas, alternating with annoying constipation. An increase in the body temperature is not uncommon.

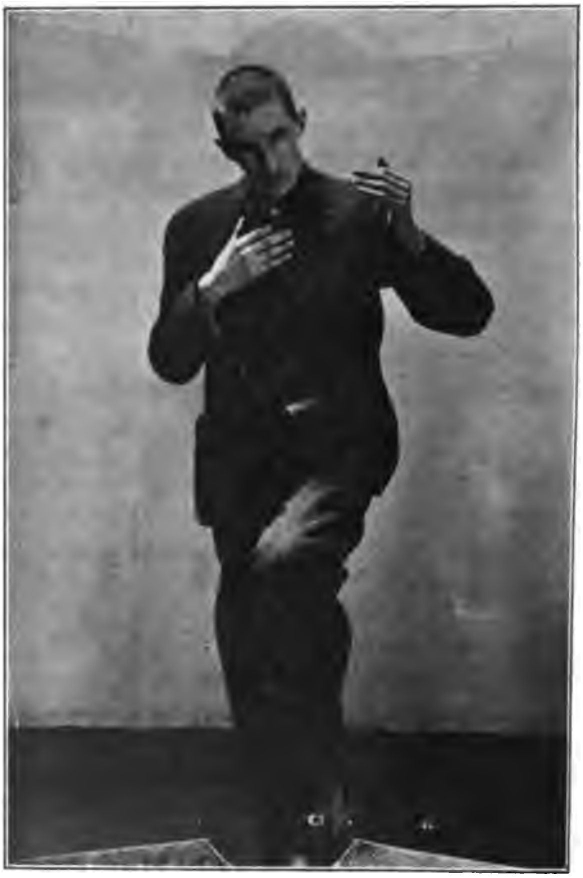

FIG. 2 CASE 2. G. B., Katatonic type.

Case report 2—G. B., aged nineteen years, single, was admitted September, 1900; laborer by occupation. No history of insanity in his ancestors. Father and mother were natives of Virginia; no particulars were furnished in regard to them. The patient received for only a few years a common school education. The history prior to admission to the hospital was most unsatisfactory. The date of appearance of the first symptoms was unknown and their character entirely obscure. Since he had been under observation, the symptoms progressed rapidly, but with almost no variability.

The patient was of dark complexion, 5 ft. 7 in. tall, and weighed about 150 lbs. All physical findings at date of entry were recorded as negative with the exception of an irregular pulse and exaggerated knee-reflexes. He was a pernicious user of tobacco. No history of past venereal diseases was ascertainable. In 1900, the patient possessed a good degree of attention to wants and surroundings. His memory loss was not marked; it was noted as fair for recent and good for remote events. He appeared constantly depressed.

At no time did he manifest delusional or hallucinatory symptoms. He had no special hobby; showed no disposition to injure others or himself; gave no display of temper. Although very slovenly and untidy in his person, he was free from filthy habits. He was inclined to pace up and down his hallway for hours at a time and his several attempts at escape implied a greater degree of cunning than his general conduct and mental attitude indicated.

A few months after admission, he had for weeks at a time strenuously resisted all efforts to feed him. Such periods ended abruptly and during the intervals of submission, he indulged in all degrees of gluttony. The first tendency to peculiar postures and stereotyped actions together with marked twitching in the facial muscles was noted about two years ago. When I saw him in 1904, well-marked muscular rigidity was present in every move he made. He remained in any position I put him; one arm down and the other held high and fixedly over his head for hours at a time was with him a popular performance. It was while in some such grotesquely statuesque attitude that I talked with him. "What is your name?" "I'll get out of the road." "Where are you?" "I can't if I know and hide; it killed me enough to get out of the road; get out of the road it's killing me," and so on without end. The voice was monotone and high pitched and emotional display was entirely lacking. He repeated mechanically the same thought over and over again, adding every now and then a cuss word such as "Damn." During the asking of more questions, he sat motionless and dumb. The facial twitching was easily noticeable around the mouth, but his gaze was fixed and the snout-cramp pronounced. After an interval, the question "Who is this?" (pointing to the house doctor.) Ans.: "I tried all I could to get out of the road. Knows better than to hurt me." (Repeating phrase four times.) Then followed a volley of terrible oaths in most excitable tones. I was told he had been in his present katatonic state for three months.

On physical examination one of the most conspicuous symptoms was the vasomotor disturbance, so commonly found in advanced or terminal dementias. The hands were of a bright red color and icy cold, but blanched to a dead white with every impression of the examiner's fingers. The pupils were widely dilated and reacted normally to light and accommodation. The knee-reflexes were greatly increased. In spite of a voracious appetite, the patient was extremely emaciated, and altogether filthy in his appearance.

In reflection of this case, there can be little doubt of its conformity to the katatonic form of dementia præcox, although data bearing on the first symptoms and their earlier development are lacking.

Dementia Paranoides—In Kraepelin's last edition of clinical psychiatry issued in 1904, some twenty pages are devoted to the consideration of a paranoid form of dementia præcox. The criticism might be ventured that much appears in this chapter that has been touched upon under foregoing headings. Some of the general symptoms and not a few of the varying psychic states, which bear such striking resemblance to those described under hebephrenia and katatonia are here referred to with such particularity that broad distinctions are lost sight of and the clinical picture is in imminent danger of being blotted out of recognition. Even Kraepelin's admirers who have given unstinted praise to his analyses and regarded his classifications as eminently progressive and rational are not altogether in accord with his construction of this type. In 1898 Aschaffenburg[37] ("Die Katatonie-Frage," *Allg. Ztsch. f. Psych.*), another staunch ally, expressed his disinclination to accept Kraepelin's dementia paranoides as a type of dementia præcox, because the material he had been privileged to see was neither sufficiently large nor long enough studied, to arrive at positive conclusions.

Aside from the presence of a rapidly progressive dementia, the delusions, illusions and hallucinations often obtrude as the initiating and foremost symptoms in the course of this form of the disease. In mode of onset, this form may prove very like the preceding ones described, wherein headache, malaise, sleeplessness, disinclination to work, mental irritability and bodily unrest were the chief complaints. It is quite usual for symptoms of excitement and agitation to come to the forefront suddenly, and so it happens entirely without warning that patients talk incessantly, in a peculiar strain, exploiting a wealth of insane ideas, divulging facts founded on intrigue against them, announcing themselves as the victims of deeply laid designs, the pitiable objects of divine wrath, seeking deliverance in long and oft-repeated prayer. Mental pictures arise in these cases which are truly weird and terrible. The auditory hallucinations of true paranoia are duplicated to a nicety. Everywhere voices rise up; they ring through the sidewalls, partitions, floors and ceilings. The visual sphere creates constant turmoil through its perception of ghosts, bloody heads, deformed bodies, and terrorizingly ugly corpses. Wild excitement seems to possess the sufferers and leads them into most quarrelsome situations. They laugh and cry by turns; and fits of unbridled hilarity continue in paroxysms for days at a time. In strange contrast, the sense of orientation as a rule remains intact, but even in their known surroundings, patients at times contemplate strange and destructive deeds such as incendiarism, personal assault and suicide.

Kraepelin cites cases of horrible infanticide and tells of a woman, who, seeing her husband asleep, imagined that he was on his deathbed and hastened, what she took to be, the desired end, by deliberately murdering him. Sad, depressive and anxious frames of mind may be short-lived and replaced by an expansive stage, in which insane delusions crowding more and more to the front, round out the entire psychic content. Exuberant in spirit and gifted in talk, they are aided by an imagination that leads them into marriages with royalty, invests them with majestic power, clothes them with the authority of the Papacy and even the Deity, domiciles them in the strangest parts of the earth and makes them omniscient. Such excessive megalomania reminds one forcibly of the grandiose, bombastic vagaries of a paretic. When enthralled by persecutory ideas their sufferings are not indicated as in genuine paranoia by expressions of fear and cringing, so much as they are exposed with beaming countenance and in a radiantly happy mood. All they require to start their expansive prattle is an encouraging word from a single listener, and the thoughts come fast and furious in long drawn out, elaborate sentences. Interruptions or adverse criticism of their incoherent fantastic talk puts them in a fearful rage.

Physical symptoms are seldom noted except perhaps those of vasomotor origin, for on very slight exertion, the skin may take on a vivid red hue. Only when a poison theory is entertained, does the appetite seem much disturbed. Sleeplessness occurs; the body weight fluctuates, but often in favor of an increase, giving these patients a ruddy and robust appearance.

The rapidity with which dementia paranoides goes on to confusional weak-mindedness, varies from a few months to a few years and despite its completeness the patients exist for as many as ten years or more, mental wrecks in a purely vegetative state.

Kraepelin has gone deeply into the recesses of this paranoid group and extracted from it a sub-type characterized by insane delusions, which are more coherent than those described, retentive for a greater number of years, but finally highly confused. These cases were cited by Kraepelin in his earlier works as "phantastic forms" of true paranoia. Of this departure he says, "whether this refinement will hold, the future alone can tell." Some authors are reluctant to call this other than a clinical variety of true paranoia, but Kraepelin for the present thinks that chronic delusions fraught with ideas of persecution and megalomania are identical with the type described and called by Magnan "délire chronique à evolution systematique" (and by Möbius called paranoia complete), and properly belong with the form of dementia paranoides and not with genuine paranoia. The paranoid form is the most unpromising as to recovery of any of the three groups.

Case Report 3—H. Z. is a young man twenty-six years old; 5 ft. 8 in. tall and weighing about 125 lbs.; single, a native of Germany. He served his apprenticeship at cigarmaking but has never found steady employment at his trade.

Family History—Referring but briefly to his heredity, it may or may not be significant to state that the patient's father and mother, both natives of Germany, were first cousins, whose respective ages at the time of the patient's birth were forty-one and thirty-four. The father died early in 1904 of some form of heart disease. It is asserted that the parents had never been addicted to excesses of any kind, and close inquiry elicits the fact that so far as any one knows, this is the first instance of mental derangement to be recorded either in the immediate family or its remote branches.

Personal History—In both his childhood and young manhood days, he is said to have deported himself in a perfectly normal way. At the age of twenty-one, he betrayed an inordinate craving for an "education," which he immediately proceeded to gratify by entering a public night school and at a later date matriculating in a business college. Prior to the advent of this apparently sound ambition, he had devoted himself to the acquisition of his chosen trade, that of cigarmaking, which, however, he deserted time and again without good and sufficient reason for temporary minor jobs of all sorts. The foretaste of education received in the night school led him to remain at the business college about three years, during which time he studied very hard and pored over his books until very late at night. In the course of his studies, he developed an extravagant admiration for renowned men, which led him to go deeply into the study of their lives and made of him a veritable hero-worshiper. Failure to pass the prescribed examination for graduation, afforded his parents and friends the first clue to the mental breakdown. He was now twenty-four years old. Following upon his failure "to pass" came a second disappointment in the form of inability to get a suitable office position.

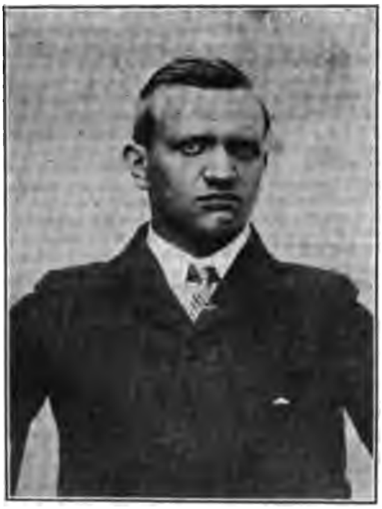

FIG. 3, CASE C. H. F., aet. 26: Dementia paranoides (paranoic form of dementia præcox).

His family believed that these two factors constituted his chief source of worry and served to establish the mental disease.

Present Illness—If his unusual conduct incident to his student days is to be reckoned with at all, it may safely be said that mental deflection was gradual in its onset, and its more thorough exhibition manifested itself some months before the commitment papers were executed.

The first series of outbursts have been extremely egotistical—his frequent allusions to himself as "the authority on familiar subjects," and exalted ideas of his own importance and ability were forced on the notice of all coming in contact with him. He claims to commune with spirits in another world, who endow with hypnotic power, and he himself is now under such a spell. His moods are variable;—there are periods when he is docile and even indifferent to his surroundings. It requires but little to animate him into a fluent yet incoherent attempt to explain his present situation. He is slightly deaf—inclines his ear, carefully intent upon getting your question well in mind—then begins his answer in a high-pitched rasping voice, which is incapable of much modulation despite the increasing excitability. The context of this rapid-fire speech is something as follows—"I testified by innocent conspiracy to give me liberty by the prophecy already at hand, foretold that I will have trouble here in law no reason being for it, that the law should kidnap me in the institution at Illinois, Elgin." Asked why he is here, he repeats the same phrase. Later interrogation on some other point elicits a sane answer like Yes or No and then again a duplicate of the above text is uttered. If an interview along these lines continues for some time, say over ten minutes, the patient is pacing the floor, gesticulating freely in the hope of emphasizing every point he seeks to make. Once after considerable cross examining, he worked himself into a veritable frenzy—and with outstretched arms invoked the aid "of some one in the skies to adjust the great wrong in the conspiracy."

A physical examination after an outburst of this kind showed equal and wide dilatation of both pupils and actively increased tendon reflexes, especially the patellars. He has hallucinations of hearing occasionally and delusions as well. His age at the time of onset, his persecutory ideas and his sense of expressed fear at some conspiracy, his constant allusion to intrigue, his high degree of a positive emotional tone, the megalomania, auditory hallucinations, incoherency of speech all typify not true paranoia, but the paranoid form of dementia præcox. Divorced from this paranoid group of symptoms, there remain the features characteristic of the dementia præcox. Orientation is fairly good. The patient knows except at most excitable periods where he is; he knew the doctor and orderly in charge of him.

This case aptly illustrated a trite distinction between attention and interest that Kraepelin dwells upon in his original article. The patient's attention could be readily arrested and he would take quick notice of things pointed out for him, but there was an absence of interest through a lack of inward stimulus to concentrate on anything; he knew what was going on but made no effort to appreciate or construe it. When his agitation ran high, he proved unsatisfactory to memory tests. His judgment was equally impaired. Stereotypy was present.

Case Report 4—E. G.; female, aged twenty, single, no occupation; father and mother were natives of Ireland. The paternal grandmother suffered from senile dementia. One aunt is now demented. The patient's father had always been eccentric and never very successful in any of his many business ventures. An uncle is said to have been "peculiar," to a degree approaching insanity. One brother who sustained a head injury when young is now an epileptic.

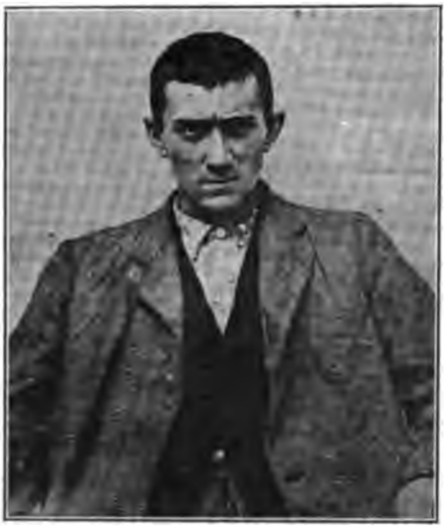

Fig. 4. Type illustrative of features and expressions often seen in the institutional cases of dementia præcox.

The patient enjoyed a good education and evinced that intense interest in her work characteristic of successful students. Two years ago (at age of seventeen), her "accumulated nervousness" consequent upon over-zealousness at the University, vented itself in a mental storm, which made sanitorium treatment at Lake Geneva necessary. I could glean from the patient's remarks that she must have been mildly maniacal at the time. A complete

A new marriage and another pregnancy were claimed. She talked incessantly. This attack at an end, she became quiet and remained docile and tractable for some months.

In this condition a physical examination of the patient, who was about 5 ft. tall and weighing 101 pounds, revealed nothing of new import. The pupils irregular in outline presented hippus activity, and were normally reactive to light and accommodation. The knee reflexes were exaggerated. In conversing, she found it extremely hard to follow me. The same answers were given to different questions. Interruption of ideas was noted. She answered grave questions with a "silly laugh" and irrelevant "prattle." Through her conversation, she sought to impress with her extreme politeness and betrayed slight effort at mimicry. In the brief interval between questions, she would repeat the last answer either all or part of it several times, thus: "Do you like it here?" "I shall go home—shall go home—when—shall home—go home." She knew where she was, remembered her physician's name; she did not recall many occurrences and as for an answer involving some thought, she proved purely defective.

Pathology—Despite the splendid efforts of such students as Alzheimer,[38] Nissl,[39] Voisin,[40] Ballet,[41] Deny,[42] Kiernan,[43] Hoch,[44] Meyer and others of note, the field of gross and microscopic pathology has proven quite sterile. Voisin, Alzheimer and Nissl have found pathologic changes in the interstitial network as well as in the cellular tissues of the central nervous system. Ballet of Paris has demonstrated anatomic sections from a case of katatonia, in which the large pyramidal cells were shown to be especially affected. Alzheimer is reported by Kraepelin to have investigated the brains of several individuals, whose katatonic course came to a sudden end with delirium, collapse and death. Here Alzheimer found an atrophic, shriveled up, cell-body, large nuclei, the extra-nuclear membrane in folds and an unusual arrangement of the glia around the cell. In 1877 Kiernan (*American Journal of Insanity,* Vol. XXXIV, p. 59) published the microscopical findings of a case and concluded: "There is a marked increase of the nuclei of the neuroglia. The ganglion cells, both pyramidal and fusiform, were normally contoured, processes well developed, protoplasm healthy, in some cases diffusely pigmented and nucleus round and clear. Free lymphoid bodies were accumulated in the pericellular spaces."

Dunton[45] (*American Journal of Insanity,* 1903, 427) reported a case of the katatonic type with autopsy. The patient had been under observation for almost four years and finally died of tuberculosis. At the outset of the paper he alludes to the frequent association of katatonia and tuberculosis and credits Kiernan with emphasizing their coexistence more than twenty-five years ago. In the amount of alteration to be noted, Dunton's case differed not at all from those studied by other investigators. A summary of the microscopical findings of the brain in his case is as follows:

(a) Only a slight cell change evenly distributed over whole brain; not restricted to any one area; (b) first frontal convolution shows greatest amount of cell change; (c) cells show central chromolysis; (d) there is slight cell atrophy; (e) atrophy, swelling and dislocation of the nucleus; (f) an endo-nucleus; (g) folding of the nuclear membrane; (h) deeper layers most affected; (i) motor cells show very slight similar changes; (j) neurogliar nuclei are slightly increased; (k) phagocytosis is well marked, also cell disintegration; (l) neither medullated fibers nor vascular structures show changes.

Deny[58] (Paris) reports the findings of a mild lymphocytosis noted by Lhermitte and Camus and adds that the blood-findings are otherwise not characteristic. In this country, W. Prout has interested himself in the bloodfindings of dementia and remarks that in the early stages of some typical cases, there was marked hyperleucocytosis and in others a decided diminution; the differential count showed a similar discrepancy.

Prognosis—Conceding mental deterioration to be the end product in all forms of dementia præcox, it is interesting to note the diversity of opinion regarding the prognosis. The hebephrenic form despite its insidious onset and more prolonged mild course seems to progress more unswervingly toward mental dissolution than the katatonic type. As stated in hebephrenia, 75 per cent. go on to profound dementia and about 7 per cent. remain moderately defective and 8 per cent. may be counted as "practical" recoveries not perfect ones, since they never acquire more than a fair mental balance, never gain a complete mastery of situations and lack in initiative force. Katatonic patients seem to fare better than this if the material governing the figures is to be relied upon.

Kraepelin holds that 59 per cent. of the katatonics end with considerable, almost complete dementia; 27 per cent. attain to a moderate degree of it, and in 13 per cent. there are "practically" no relapses or residuals. Some writers have expressed themselves rather optimistically in regard to the prognosis of the katatonic cases, animated thereto by the knowledge of long lucid intervals, but other alienists with vast material at their disposal are less inclined to be even sanguine, and Kraepelin feels justified in giving an unpromising prognosis for the reason that after five, seven and ten year lucid periods intervene, the morbid mental state has been known to reassert itself.

Norbury[46] furnishes the data of a case (*Lancet Clinic,* January 7, 1905) of katatonia in a student nineteen years old, who passed through the stages of stupor and excitement and "finally made what was a complete recovery," since for 15 years he has not shown any mental disturbance and is now a prominent and successful farmer. This case might well be credited with recovery, but one cannot help reverting to the Kraepelinian text on long remissions in which is substantially stated, that a great series of his apparently cured katatonias relapsed, leaving the author unable to decide the percentage of "recoveries" in the fullest sense of that word. That the prognosis of the katatonic type is regarded as more promising than the hebephrenic is by some writers attributed to the acute onset and more active brisk course assumed by the former. The forerunners of death in these cases are chiefly exhaustion and collapse from sustained grave excitement, tuberculosis and injuries or accident.

Differential Diagnosis—The differentiation of psychic diseases is neither easy nor profitable, nor of absorbing interest without the presentment of carefully annotated and elaborated case histories, but the gross points of similarity and dissimilarity between dementia præcox and circular insanity, paranoia and general paresis, surely merit more consideration than most writers have accorded them. For many it is quite enough to know that a case is one of insanity and that the prognosis is favorable or unfavorable, while clinical distinction seems for the most part to be irrelevant.

Dementia præcox requires to be differentiated chiefly from circular insanity, especially at the time of the first attack, which so often initiates itself in adolescent life. Aschaffenburg submits figures regarding this point as follows: In 127 cases of circular insanity 72 per cent. experienced their first attack before the 25th year; in the males diagnosed as præcox cases 74 per cent were affected before the 25th year and of the women 80 per cent.

Depression is common to both diseases in the very beginning, but the calm resignation in the circular cases originates in inhibition or what the Germans call "Hemmung," whereas the inaction of dementia præcox results from negativism. In consonance with this fact, the movements carried out by circular cases are slow, tardy and dilatory while in dementia they default altogether or are contrarily executed. Meyer in his "Review of Recent Problems of Psychiatry," observes relative to the free use or perhaps abuse of the word Hemmung—"as especially pleasing to me, I refer once more to the simple statement that effective melancholia consists in depression plus intrapsychic akinesis, instead of what Ziehen and Kraepelin speak of "Hemmung" or inhibition, a term too easily used and apt to suggest a deus ex machina. The simple description, akinesis or retardation, is decidedly more objective."

Gross, with a view to ascertaining the reactionary power in these cases, studied the finer phases of coördination displayed in hand-writing and concludes that melancholiacs form small letters writing slowly and evenly, exerting but little pressure, while the stuporous dements write unequally large and small, slowly and rapidly and without regard for uniformity in tracing similar letters. The mixed forms, so-called manic-depressive states, present great difficulties.

Concerning paranoia, to which weak mindedness is absolutely foreign and in which the gradual development of a systematized delusion is insisted upon as vital to the diagnosis, there can be little chance for error in differentiating this disease from dementia præcox, but where insane delusions occur, of a hazy, disconnected and unelaborated type and the intellect suffers a let down and setback betokening progressive dementia, a proper estimate is at once beset with difficulties. In such cases, the tics peculiar to dementia præcox may clear up a doubt, for they play no rôle whatever in paranoia proper. It has been said of general paresis that it resembles dementia præcox closely even to the verbigeration and negativism; of the latter it may be said that it is accidental to general paresis and incidental to præcox. Katatonic symptoms are frequent in general paresis.

Putting aside consideration of the reflexes, the pupillary anomalies and motor symptoms, the chief diagnostic difference after all rests with the mental state,—in paresis the memory defect early and grave,—in dementia late and wild. Minor conditions open to question in diagnosis are congenital dementia, imbecility and idiocy.

An interesting case studied by Kaiser[47] (*Allg. Zeit. f. Psych.,* 1905) has led him to write it up under the title "Dementia Præcox or Brain Tumor?" in which he says he felt compelled from the mode of onset and symptoms to ask himself the question—Is this case one of dementia præcox with the accidental development of a brain tumor, or is it a case of brain tumor masquerading in the guise of dementia præcox? The post-mortem diagnosis read, "Gliomatosis of the right hemisphere, with hemorrhages and areas of softening; pachy- and leptomeningitis of the whole convexity of the brain." The patient was a female, with a bad heredity, who enjoyed good health to her twenty-second year and then experienced the first mental storm of hallucinatory type, violent and sudden in onset. She remained in this state from 1892-1902; then there appeared attacks of Jacksonian epilepsy and an advised operation was not consented to; death followed shortly thereafter.

Dementia Simplex—In a masterly and exhaustive paper ("Ein klinischer Beitrag zur Kenntniss der Verblödungspsychosen: *Archiv. f. Psychiatrie und Nervenheilkunde,* 1903). Otto Diem[26] has asked for the recognition and acceptance of another clinical type of Dementia Præcox—"Die einfache demente form" (the simple demented form; dementia simplex), which is not to be confounded with same term applied by Rieger, who preferred it to the appellation Dementia Præcox. Perusal of his nineteen carefully prepared case reports compels more than mere passing acknowledgment of his effort to further amplify the existing classification. His attitude is best deduced from his own summary, which translated reads:—"Besides the recognized clinical types of hebephrenia, katatonia, and dementia paranoides, all of which end in a peculiar and special kind of mental weakness, there is still another closely resembling these in point of altered disposition, intellect, and termination. In this form, the onset is always without prodromes, simple and insidious; the course without acute exacerbations and remissions; without maniacal or melancholiac moods; without insane hallucinations or delusions and minus such characteristic peculiarities as the tics, mannerisms, stereotypy, negativism, mutism and cataleptic phenomena."

Pure cases of this type are rarely studied for the reason that they may never come under observation, unless for a short time only and most of them are well advanced before they are committed to asylum care. Diem contends that the heboidophrenias of Kahlbaum and Weygandt as well as the primary dementias of Sommer are but transition forms to genuine hebephrenia—not meriting separate clinical preferment, since with course, prominent symptoms, and termination alike in both, the differences are rather those of degree than of kind. In his first twelve case citations, all specifically hebephrenic symptoms throughout the entire course of the malady have always been conspicuously absent, which fact alone predisposes to the creation of a new type. In conclusion, it is remarked that neither heredity nor puberty stand in wholly adequate causal relation to this form, since several cases are recorded in whom the antecedents were without taint and the disease inaugurated after the thirties. Women afflicted with this clinical form are frequently countenanced in the body-social as bad characters, and men simply as alcoholics. The intricate relationship this mild psychosis bears to states of alcoholism and conditions of vagrancy gives it a practical and forensic importance not to be underestimated. Willmanns'[48] (*Neurolog. Centrbl.*, Dec. 16, 1902) inquiry into tramp life also confirms this reflection. The material reported consisted of 120 psychoses sent from Kislan penitentiary to the Heidelberg asylum, and it will suffice to say that 66 (6 females and 60 males) were cases of dementia præcox.

Critical Review—Leaving the devious yet well beaten paths of technical discussion, it should surely prove of interest to know with what critical temper the present devotees of psychiatry have accepted or rejected the Kraepelinian doctrine, if such it may be called. In a retrospect of this phase of the subject, it is necessary to turn back farther than the year 1903 for disquisitions of approval and disapproval, utterances conservative and radical emanating largely from German, French, English, and American sources. It must be noted that in some instances good papers have paved the way for poor discussions; and many of the contra-contentions seem borne of an unknowing, irreverent or almost vicious frame of mind.

Kraepelin's choice of the term dementia præcox has been responsible in large part for the early confusion, shared by alienists the world over. This appellation had figured in French literature as Christian's[49] demence precoce, merely another name for imbecilitas tarda; Tschisch had used it in a similar sense; Bernstein[50] tells us that "Demence precoce des jeunes dégenerés and Ansemoff's præcox" embraced but a small percentage of the cases (chiefly congenital in origin) that Kraepelin sought to cover. Serbsky's[51] notion of this psychopathic state, differs from others in that he regards it as not always a primary disease (ein originäres Leiden) but one frequently following the acute psychoses and as such becoming "dementia secondaria progressiva." Some writers have never been wholly reconciled to the present broad far-reaching declaration, which has in effect swept their preconceived hobbies out of the way.

From the very first, English authors have taken exception to the modifying word "præcox" in the title, which, implying "youth," seemed much too inelastic a term for an affection often beginning in the third and fourth decades. Recently the question has been asked whether all early dementias except those of general paralysis, epilepsy, alcoholism are to be included under dementia præcox. More cause for dismay arose from the changes made by Kraepelin in the editions succeeding the fourth of his Psychiatry; in the earlier issues he retained the term dementia præcox as interchangeable only with that of Hecker's hebephrenia, and later extended its meaning to cover all the groups.

In the eyes of some alienists another offense appeared in Kraepelin's habit of speaking of "Katatonics" *per se* (Katatoniker) long after he had subordinated them to mere types of dementia præcox. The fact that Kahlbaum, Schüle,[52] Tschisch, Korsakoff, Serbsky and Kraepelin (in 1896) had all advanced "katatonias" of their own creation, served to increase the disadvantage under which students in this field were working.

A glimpse at the current literature will readily convince the student with a liking for psychiatry that dementia præcox has been in the limelight of continental medicine for some time. Not alone have the smaller special societies from time to time aired this topic; it has been for the past years the elective theme at the larger European Congresses. I have in mind the reports of the Section on Psychological Medicine in England (*Brit. Med. Journ.*, Oct. 15, 1904) and those of the Convention of French Psychiatrists and Neurologists (*Revue Neurologique,* Aug., 1904). both of which afford ample proof that the subject is admittedly the fashionable one of the hour on the Continent. Of the French psychiatrists, Deny,[52] whose paper on "Des demences vesaniques" it was that animated the frank discussion, and Brissaud,[53] who presided over the congress, showed a kindlier feeling for the Kraepelinian doctrine than did Parant, Vallon, and others. In fact Deny felt constrained to resent the accusation of Germanomania lodged against him for a too pro-Kraepelinian attitude. Deny's contentions were in substance, that whereas dementia præcox had been conceived on French soil by Esquirol and Morel, the honor and credit belonged to Germany and her Hecker, Kahlbaum and Kraepelin for breathing a new and lasting life into its frail form. He agrees that the psychosis is capable of division into the hebephrenic, katatonic and paranoid forms and constitutes perhaps 25 per cent. of the asylum cases, but discards the notion of secondary dementia and secondary insanity as having anything in common with the above mentioned forms. He does not regard this psychosis to be of pure degenerative origin, and as for Dupres'[53] attempt to establish the atypic causes as "Pseudo-dementia" he holds this to be most premature. In closing Brissaud remarked with much truth that more misunderstanding of Kraepelin's teaching had arisen from generalizations couched in an apodictic style by his followers, than through any fault of his (Kraepelin's) own. Regis,[53] of Bordeaux, chose to be an opponent of the newer German terminology and warned against the French acceptance of dementia præcox as a disease entity. In his opinion, there could be but two (2) groups with a constitutional basis, one the hebephrenic and another embracing hallucinatory insanity, which might recover or go on to secondary dementia; only the hebephrenic forms could be construed as dementia præcox and in this sense had long been understood by Morel. Parant,[50] Crocq,[51] Garnier,[52] Colin[53] and others discussed minor features based largely upon their own observation. So much for the French spirit.

The best résumé of English thought comes to us through the published discussion of a paper read in England by Conolly Norman[54] on dementia præcox. In this critical paper, the forms of hebephrenia and katatonia are gracefully accepted as distinctive enough to merit such appellation, but of the paranoid forms he adds: "There is absolutely no criterion whereby we can distinguish these from paranoia unless we suddenly drop the method of inclusion which we have been hitherto following and, reverting to the method of exclusion, hold that nothing can be called paranoia except the most exquisite pure and typical cases." Just what Norman means to say in this phrase is not clear, and therefore fails to excite more than idle curiosity. He takes exception to the origin in adolescence and to many others of the assigned causes; he deprecates the use of both terms "dementia" and "precocious," and takes issue on other points too numerous to mention, but he does add by way of compliment, "It needs here to be again pointed out that Kraepelin does not claim as much for his precocious dementia as do his followers. He expressly states that the existence of dementia præcox as a separate and distinct disease is doubtful and he points to the prognostic advantage of stringing together what may later on prove to be separate affections by the common thread of their tendency to dementia." Throughout the essay there are partially successful feints at magnanimity, but in the closing paragraph is faithfully revealed the uncompromising tenor of English psychiatric thought, which reads: "Classification is not a very important things in the existing state of psychiatry except for convenience in teaching, and the mere nomenclature of disease is of little value save as an index of our knowledge or ignorance. Taking note, however, of natural groups is of vast importance with a view to prognosis and treatment, and it is therefore safer and more scientific to have no classification than one which confuses together under one denomination groups that have no natural connection, more particularly when such classification tends to establish counsels of despair." Dawson,[54] White,[54], Bower,[54] Shuttleworth[54] were in thorough accord with Norman's exposure of the fallacies of Kraepelin's work.

About one month prior to this English arrangement, Dercum[80] brought the salient, debatable features of dementia præcox before the Section on Nervous and Mental Disease of the American Medical Association, at its fifty-fifth annual session (June, 1904). He too objects to the use of the term "dementia" for reasons "not only scientific but ethical." To him its very sound conveys an unfavorable prognosis and he greatly prefers to employ such terms as Das Jugendirresein or its English equivalent, Insanity of Adolescence. He adds: "This presupposes nothing as regards the future and leaves the question of recovery to be determined by the course of the case." When Dercum seeks to deprecate the value of the term "hebephrenia" and finds a better one in his designation of a "simple expression of dementia præcox," he fails to reckon with the class of cases described originally by Kahlbaum as Heboidophrenia, later called by Weygandt, dementia simplex, and within the past two (2) years exploited by Diem as dementia simplex (die einfache demente Form). According to these writers, there are forms simpler than the simple one, which Hecker described and Dercum would conveniently dispense with. It is only too true that matters would be facilitated by alluding to a simple, katatonic and paranoid form of dementia, but if the term hebephrenia *per se*, conveys according to Hecker and all other writers one idea and dementia simplex another they are not interchangeable, and Dercum's effort to simplify the terminology could only be sanctioned if all observers were a unit in doing away with finely drawn, confusing distinctions.

Dercum finds it unpleasant to reflect on the plan of incorporating with dementia præcox, the phantastic forms formerly classed with paranoia and says, "I cannot but feel that this change has been an unfortunate one, and that by it Kraepelin does violence to facts." The entire arrangement by which adult cases of paranoia are grouped with those of "dementia præcox" is distasteful to him and he suggests in his closing of the discussion "that it were better to classify the juvenile cases under the term insanity of adolescence and leave off the term dementia præcox."

Enough time elapsed between the months of July and October, 1904, to allow for the wave of "præcox" criticism to reach American shores. The New York Neurological Society recently entertained its members with a symposium on dementia præcox, viz.: Has the recognition of dementia præcox advanced our conceptions of the various forms of mental derangement occurring in the adolescent period? Is not the present tendency to give too grave a prognosis in the youthful insanities? How frequently does dementia follow the mental derangements of early life? I am not familiar with the full text of the papers read on this occasion, but the opinions voiced in discussion by Meyer,[55] Sachs,[55] Dent,[55] Hirsch,[55] Collins,[55] and others may prove of interest as reflecting more of the American view. Adolph Meyer whose research studies in experimental psychology and clinical psychiatry have contributed so much towards standardizing American thought in this field, whose word and argument is capable of creating a firm prejudice, acknowledged at the outset that the issue was a far-reaching one. That Kraepelin in the fifth edition of his book had done away with katatonia and hebephrenia as degenerative conditions and granted them an acute onset in apparently sound individuals on an autotoxic basis, had filled him with wonder and surprise. This new pose of the Heidelberg school he felt was "due to controversial reasons rather than to actual conviction." He felt convinced that Kraepelin's writings had induced psychiatrists to break away from the old traditions of classifying all forms of insanity into melancholia, mania and paranoia, thereby neglecting well-marked and striking forms of mental disease, which could be readily recognized. He added without hesitancy that Kraepelin had gone too far, a fact that he himself (Kraepelin) admitted and sought to apologize for in submitting the classification for furthere revision. Meyer thinks that the cases in which all symptoms are not present may still show signs of deterioration and should be regarded as allied to dementia præcox. Whether this implies the recognition of a "pesudo-dementia," is not stated.

In making the statement that the term dementia præcox should be carefully restricted to cases in which mental deterioration is recognizable at an early stage and not anticipated for a far distant future, Sachs emphasized a point worthy of most careful consideration. His remarks on the injustice of immediately pronouncing a precocious dement as incurable are well placed, since it is conceded that so very many individuals afflicted retain enough psychic balance to render service for themselves and others in the body social. To offer a more encouraging prognosis than Kraepelin thought it possible to give, seemed to him feasible. Sachs is of the opinion that little good can come of grouping widely divergent types under one heading and favors a return to the older plan of clinical subdivision.

SUMMARY.

1. Heinroth in 1818 hinted at various forms of dementia and in one of these perhaps anticipated later descriptions of dementia præcox.

2. From the time of Esquirol (1838) to Rousseau (1857) French labors in this field ceased but were renewed from 1860-1886 with vigor by Morel, Legrand du Saulle, Falret, Legrain, who aimed to correlate arrested development with the pubescent age and emphasized the mode of onset.

3. The English school of psychiatrists led by Tuke and Clouston, added the weight of their authority by teaching that the essential nature of adolescent mental disease lay in the "tendency to dementia from the very beginning."

4. German interest was awakened in 1871 by Hecker, who it is believed employed for the first time the term hebephrenia and defined its scope quite as accurately as it is at present constructed.

5. Following directly in the footsteps of Hecker, Kahlbaum by subtle analysis evolved a milder, abortive, curable type, which he chose to call Heboidophrenia. Kahlbaum's somewhat later paper on katatonia was a classic. Even at the present time his katatonia is by many regarded as a clinical entity.

6. The subsequent writings from 1883-1903 of Neisser, Pick, Griesinger, Sommer, Krafft-Ebing, Wernicke and Diem

have materially advanced the knowledge and study of adolescent insanity.

7. Kraepelin recognizing the imminent need of clarifying all thought on this subject, set himself to the Herculean task of classifying anew, and submitted for the first time under a title not wholly untried, a series of disease pictures in group form, all of which had in common a termination in a special kind of mental weakness. The title was that of Dementia Præcox. The classification was a radical step in advance of anything hitherto accomplished. In the fourth edition, the degenerative psychoses were arranged in three groups, (a) dementia præcox (Hecker's hebephrenia); (b) katatonia; (c) dementia paranoides. In the seventh edition (the last), no mention is made of degenerative psychoses; the general designation of the chapter is dementia præcox, and the three groups are (a) hebephrenia; (b) katatonia; (c) dementia paranoides.

8. Kraepelin's definition and general symptomatology are briefly considered, some of the important phenomena receiving proper emphasis by way of case references.

9. The three groups fashioned by Kraepelin are separately discussed with attached short case reports.

10. Pathological research done by Alzheimer, Nissl, Voisin, Ballet, Kiernan, Hoch, Meyer has given little more than an insight into the structural changes that underlie the diseases, but the changes noted are sufficiently suggestive to stimulate to still keener investigation.

11. Despite conflicting statements it appears that the optimism concerning curability should in all instances be held in check. Most authors agree that the prognosis is even better in the katatonic form than the hebephrenic, and most unpromising in the paranoid. A large material and very long interval of freedom from symptoms is necessary to govern the ultimate dictum as to prognosis.'

12. In the differentiation of dementia præcox from circular insanity, paranoia and general paresis, signal symptoms such as the memory loss, the judgment, negativism, verbigeration, manner of speech, character of the delusions and hallucinations, the tics and katatonic symptoms, must be carefully weighed, since some of them may be manifest in all forms of insanity.

13. Diem's dementia simplex is deserving of note, since it differs from all of the aforementioned types by virtue of the following characterization: an onset without prodromes—insidious, without exacerbations or remissions—without maniacal or melancholiac moods—without insane hallucinations or delusions, without tics, stereotypy—negativism or cataleptic and katatonic phenomena.

14. The essentially controversial character of the discussions entered into by contemporary alienists of France, England and America fails to admit of abridgement here.

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The President, Dr. Joseph Fraenkel, in the Chair.

A Case of Acute Psychosis in the Course of Tabes.—This was presented by Dr. George H. Kirby. The patient was a woman, 50 years old, whose family history was negative. Her early development had been normal. She was married at eighteen and had nine children, only three of whom were living. The last child was born fifteen months ago, and died at eighteen months. It was delicate from birth; its hair fell out, and it had some eye trouble, which the doctor told her had been inherited. She questioned her husband at the time about venereal disease, but he denied infection. She believed, however, that he had been treated shortly before this for some such disease. She had never seen any signs of disease on his body. She herself had had no sore, so far as she knew. She never became pregnant again.

About eight years later, *i. e.*, in 1899, she began to have headaches and shooting pains in the legs. In January, 1903, she suddenly became exhilarated and lively, claiming that she was going to make a thousand dollars a night singing, and that she could heal the sick and perform miracles; she spent money foolishly and began to drink. During her periods of excitement she had several fainting spells, but no convulsions. About a month after the onset of these symptoms she was brought to Ward's Island. A physical examination at the time of her admission showed that the knee-jerks were absent and she had Argyll Robertson pupil; some Romberg swaying, and greatly diminished pain sensibility below the knees. There was a fine, regular tremor of the tongue and hands; no speech defect.

The main facts in the mental status were the following: She was quiet, pleasant in manner, perfectly oriented in time and place; she answered questions freely, was connected in speech, but tremendously expansive. She claimed that she had wonderful powers, could speak every language, and possessed a beautiful voice. On other topics the patient talked to the point, and gave a good general outline of her life. There was no sensory defect for either recent or remote events, but in her statements of certain remote dates there were discrepancies which she only partially realized, and was not able to fully correct. Her attention was good; she retained names and numbers well. Calculation was somewhat defective for anything beyond simple problems, but this was probably due to the fact that she had received very little education. The first specimen of writing, taken on admission, showed a few errors in the spelling of difficult words, with an occasional poorly-formed letter and rarely an extra stroke. Later, the writing was practically without defect. After she had been in the hospital two and a half months she rather suddenly became clear mentally, gained full insight into her previously expressed grandiose ideas, and her memory was without defect. The physical signs of tabes remained unchanged. She left the hospital on June 27, 1903.

A few months after her return home she began to suffer from pains in the back and a sensation of constriction about the waist. She had difficulty in walking and her legs would suddenly give way; or, if she bent over, she was apt to fall on her face. Her tabetic symptoms had been progressive, with no return of the mental trouble. She was now helpless so far as locomotion went. The joints were relaxed. Deep sensibility was lost in both legs. Three months ago she had an enormously swollen hip, which appeared rather suddenly and without pain; this swelling had recently disappeared.

The patient showed a wonderful amount of energy and perseverance. By crawling about on the floor and having things put where she could get them she did her own housework, cooking, washing, etc. Her memory remained good, and she retained full memory of her previous mental attack.

A Progressive Lesion of the Root of the Fifth Nerve, Producing Motor, Sensory and Trophic Symptoms.—Presented by Dr. J. Ramsay Hunt. The patient was a man, fifty-nine years old, who had been a submarine diver for forty years. At the age of twenty-five he had a chancre, of doubtful nature. His present disease began about sixteen months ago, and consisted of disagreeable drawing sensations and paresthesia on the left side of the face, in the distribution of the left fifth nerve. With this there was an associated weakness of mastication on the same side, and a tendency to a constantly chewing motion, which still persisted. Since the onset up to the present time, the affection had been gradually progressive.

On April 12, 1904, about four or five months after the onset of these symptoms, he descended 45 feet below the surface of a reservoir to extricate a fellow-diver who had been held there for forty-eight hours in the suction of a large water-pipe. Before reaching the bottom, however, a feeling of faintness came over him, and he gave the signal to be drawn up. Upon reaching the surface he was a little weak, but otherwise felt as usual. He did not lose consciousness, and returned home without noting any ill-effects from his seizure.

The next morning, on awakening, he could not see with the left eye, and an oculist who examined the interior of the eye said that there had been a hemorrhage. Quite by accident the same day, on attempting to use the telephone he noticed that he could not hear in the left ear. There was not, nor had there been, any tinnitus aurium. Within two weeks the sight returned to the left eye, and the hearing was restored to the left ear.

The symptoms in the region of the left fifth nerve increased, and were as follows on his admission to the Cornell Neurological Clinic, April, 24, 1905: Paresthesia and weakness in the distribution of the left trifacial nerve, associated with an incomplete anesthesia to touch, pain and temperature in the same area, including the mucous membrane of the eye, nose, tongue and mouth on the affected side. There was an absence of the conjunctival and corneal reflex on the left side. He had a drawing sensation in the left face and gums, a feeling of irritation in the left eye, and the left nostril did not feel free. He had at no time had pains in the left face. The sense of taste was diminished on the left side, and the sense of smell, although not very acute on either side, was also diminished on the left.

The left face was smaller than the right, and this did not seem to be entirely explained by the wasting of the temporal and masseter, which were atrophied on the left side. A large section of the frontalis muscle was wasted and did not respond either to voluntary innervation or to electricity, and the tissues were thinner than on the unaffected side. The following oculopupillary symptoms were present on the left side, *i. e.*, slight ptosis, enophthalmus and moderate dilatation of the pupil. An attempt to test the sweat secretion of the face by the administration of pilocarpin and the tear secretion by irritation of the conjunctiva were negative.

An ophthalmological examination made by Dr. J. H. Claiborne showed that the vision in the right eye was 20-30ths; in the left eye 20-50ths. The left disc was slightly pale and hazy, and a few old retinal hemorrhages were seen in the peripheral portion of the left retina.. The ocular excursions were normal, with marked nystagmoid twitches on looking to the right and left, especially toward the left. He had had transient diplopia.

The facial innervation was normal on the left side, with the exception that the upper, outer half of the frontalis muscle did not respond at all either to innervation or the electrical tests.

Electrical: The responses, both direct and indirect, to the galvanic and faradic currents were moderately diminished on the left side; no qualitative changes.

The other cranial nerves, the gross motor power, the gait and station, the tendon, skin and pupillary reflexes were all negative. He had occasional vertiginous seizures and suffered somewhat from insomnia, but no headaches.

Dr. Hunt said that the motor, sensory and trophic symptoms, without root pains, suggested a lesion of the nuclei of origin of the left fifth nerve. The nystagmus would also indicate pressure in the pons. The retinal hemorrhages must be regarded as an accidental complication. The temporary deafness, without tinnitus and with complete restoration of hearing, was more difficult of interpretation. Symptoms of hysteria were wanting. The nature of the process might only be conjectured. It was, however, progressive and unassociated with general symptoms of tumor.

Dr. Charles L. Dana said he had had an opportunity to examine the case shown by Dr. Hunt, and it was certainly a very unique one. There seemed to be a progressive facial hemiatrophy, with progressive loss of function of the fifth nerve. There was a gradual wasting of the left side of the face, with a certain amount of motor loss, including also the motor branch of the fifth. There was no pain nor symptom of tumor. The condition could best be explained by some morbid or degenerative condition involving the nuclei of the fifth nerve, possibly a gliosis or a parasyphilitic lesion. The case might eventually turn out to be an aberrant form of tabes. Dr. Dana said he had seen some cases of tabes with these peculiar symptoms, but never with a progressive facial hemiatrophy developing in this artificial way.

A Case of Lead Poisoning, Probably Complicated by General Paralysis.—This was reported by Dr. C. B. Dunlap, with specimens. The patient was a man, forty-seven years old, a painter, who was admitted to one of the State Hospitals on August 1, 1903, and died there seven months later. He had had painter's colic two years before admission, and had not been considered well since that time, though he had worked at odd jobs up to a week before entrance, at which time he left his boarding-house, was found wandering in the street in an aimless, bewildered way, was arrested and taken to the police station. A woman with whom he had boarded previous to this time said that he had talked to himself and was nervous and restless, but otherwise apparently normal. The certificate stated that he was very forgetful, took things not his own and talked in a silly way.

At the hospital he appeared to be much exhausted, dejected and apathetic. He did not know where he was, or the nature of the place, or the date, and gave the date of his own birth incorrectly. He claimed to hear a voice that he could not place.

Examination showed sluggish reaction of the pupils to light and accommodation. The tongue was tremulous. There was partial paralysis of the left arm and hand, and a much weakened grip on that side. There was apparent wasting of the muscles of the thenar and hypothenar group; this wasting was also present on the right side, but less marked. The elbow, wrist and thumb reflexes were present on both sides. There was slightly diminished pain sensation on the ulnar side of the left arm. There was apparent wasting of the shoulder muscles, more marked now on the left side; also of the back and gluteal muscles. The anterior tibial muscles showed some weakening, more marked on the left side. The knee-jerks were exaggerated; the plantar reflexes diminished. No clonus; no Babinski; no sensory disturbances.

His speech was mumbling, and so slow that it was often difficult to understand him. He said he was tired and exhausted and knew that he was sick, but did not know what was the matter. He saw "visions and spirits," and believed he was in the hospital because he "did wrong." A month after admission he thought that he had had some brain trouble on entrance, but that he had improved. In this he was correct.

In November he complained of vague pains in his legs.. The following month he became irritable and quarrelsome, and subsequently his gait became unsteady. The latter part of January he began to show marked incoördination, with great exaggeration of the patellar reflexes. The muscles of the legs, hands and tongue showed a marked tremor, and there was a peculiar trembling speech intonation, with failure to properly pronounce many words. By the middle of February the speech was worse, and the hands so ataxic and tremulous that he could not feed himself. Mentally, he was depressed and worried about himself. Four days before death the incoördination was jerky, almost spastic at times. He was almost hypersensitive to pain stimuli. Wasting of the interossei of the hands and feet and of the muscles generally was noted. All the muscles responded to the galvanic current, but the response was slight in the gastrocnemii and interossei. The day preceding his death the twitching of the muscle-groups was still more marked.

Autopsy: There was hypostatic congestion of the lungs; the spleen was soft and somewhat enlarged. The pia arachnoid was thickened and water-logged. The brain showed considerable thickness and toughness of the pia of the convexity and base. The basal vessels were fairly thin; there was no atheroma. There were no gross lesions, and on section the appearance was normal, with nothing to explain paralysis of the left arm and hand. There were no granulations in the fourth ventricle.

Microscopically, sections taken from the paracentral and frontal cortex of the formalin hardened brain showed the typical changes found in general paralysis, namely, infiltration of the pia with lymphoid and plasma cells, infiltration of the walls of the cortical vessels with the same elements and with pigment and pigment-carrying cells. There was also marked apparent increase in vascularity of the cortex; also plain neuroglia reaction, seen in the first layer of the cortex in the form of numerous spider cells; disorganization of the cortex layers, and numerous rod cells, such as Nissl and Altzheimer found to be quite constant in general paralysis, and unusual elsewhere. The walls of many of the larger cortex vessels were so packed with cellular elements and so broken up that the different coats were indistinguishable, and the lumen was often hard to find. Marchi stains of the paracentral lobules showed a few myelin sheaths, which were broken up into rows of fat droplets, indicating a myelin decay of moderate degree. In sections of the spinal cord from the cervical, thoracic and lumbar regions, a diffuse myelin degeneration was evident in the anterior or posterior roots.

This case, Dr. Dunlap said, presented many difficulties, and was shown in order to call attention to the absence of sufficiently reported microscopic examinations in cases of lead encephalopathy. Quensel, in 1902, working on this problem, stated that lead poisoning was capable of producing "all transitions up to typical paralytic findings." In the case under discussion there was no history of syphilis, no ependymal granulations in the fourth ventricle, and the history was far from typical of general paralysis. The speaker did not wish to say that this was a case of lead poisoning with the typical anatomical changes of general paralysis. It might have been a case of the latter affection, in which the plumbism was merely a coincidence.

Dr. T. P. Prout said he could recall three toxic cases resembling that reported by Dr. Dunlap, but none of them came to autopsy. The toxic symptoms that were particularly prominent were decided somnolence and a passive depression. All three of those patients came from a certain factory in Paterson, N. J., where rubber was manufactured, and the speaker said he was inclined to attribute the toxemia to the noxious gases that were generated in the factory and inhaled by the men.

A Case of Brain Neoplasm.—Presented by Dr.. William B. Noyes. The patient was an Italian boy, eleven years old, whose family and previous personal history were negative. He was one of eight children, three of whom had died at birth. There was no history of syphilis or tuberculosis. Two months ago he developed some affection of the eyelids resembling ptosis; this was noticed first on the right side, and disappeared after a month upon the application of a leech behind the ear. The left eye became similarly affected a month after the appearance of the condition on the right side. He has complained of frequent headaches, located in the region of the right ear and right frontal region.

Examination showed the following condition: A left facial paralysis of a peculiar tyne, with continuous spasm and twitching of the orbicularis palpebrarum, and blepharospasm. Reaction to faradism absent. The tongue showed choreiform movements, or a fine fibrillary twitching. There was double choked disk; no paralysis.. The temperature usually ranged between 99 degrees in the morning and 100 or 101 degrees in the evening. An internal strabismus subsequently developed, due apparently to overactivity of the internal rectus rather than to paralysis of the external rectus. The conjunctiva of the left eye was anesthetic, but there did not seem to be any other fifth nerve symptom. The left knee-jerk became exaggerated; there was some ataxia on standing with the eyes closed. The headaches recurred frequently, though yielding to ergot. The boy's general health remained good enough to permit him to play in the yard.

Dr. Noyes said the case was regarded as one of cerebral neoplasm, located in the pons, or, according to the view of one observer, in the facial center in the cortex. The speaker said he believed it was located in the pons or cerebellum, and pressing on the pons, because of the involvement of so many cranial nerves or their nuclei. The nature of the neoplasm was doubtful, excepting that the temperature strongly suggested an abscess. The symptoms, however, seemed to be too distinctly limited to make an abscess probable.

Dr. William M. Leszynsky said that one of the interesting peculiarities of the case shown by Dr. Noyes was the left facial spasm, a certain degree of which was sometimes noted after a facial paralysis in older patients. From the history of Dr. Noyes' case, the speaker said he would be rather led to infer that the lesion, instead of being an actual neoplasm, was probably a disseminated one, or that the symptoms were due to a certain amount of basilar meningitis. It was hardly necessary to assume the presence of a neoplasm on account of the extensive choked disk, and he doubted whether any ophthalmologist who looked into the eye and found hemorrhages and choked disk could make the diagnosis of a neoplasm in contradistinction to a basilar meningitis.

Incomplete Transverse Myelitis from Exposure After Working in Caisson.—Reported by Dr. I. Abrahamson. M. G., male, a Russian laborer, came under observation on March 23, 1900. His family history was unimportant.. Seven years ago the patient had typhoid fever. He had long been addicted to the excessive use of beer and cigarettes. Denied venereal infection.

Up to fourteen months ago he worked for three hours daily in the Pike Street caisson. For three days previous to the onset of his present illness he had been dissipating and drinking heavily.. He entered the exhaust-chamber feeling ill at ease, and remained there twenty minutes; while there he drank some hot coffee and then went out into the rain without adequate protection, and reached his home soaked and cold. About ten minutes after leaving the exhaust-chamber he began to vomit, and was seized with violent pains in the epigastrium. He felt cold, with chilly sensations and numbness in the legs, especially the left. At the same time there was increasing weakness in the legs, so that within fifteen minutes after going to bed he was completely paralyzed from the umbilicus down. There was also retention of urine and feces, and marked dyspnea and palpitation. He was taken to the Gouverneur Hospital, where an examination revealed the above train of symptoms. After a month or so he slowly began to improve, but he still suffered from constipation and delayed urination. The gait was very spastic, with slight unsteadiness.

The lesion in this case, Dr. Abrahamson said, was an incomplete transverse myelitis. Etiologically, the case illustrated the importance of alcoholism and exposure as predisposing to caisson myelitis, facts which numerous writers had already accentuated.

A Case of Syringomyelia.—Reported by Dr. Abrahamson. The patient was a Hungarian, 42 years old, a tailor by occupation. He had five living children; one child had died of measles. His wife had never miscarried. His father died of tuberculosis, his mother of gangrene of the leg. He had five living sisters, who were all well. His family history was otherwise negative. The patient stated that he suffered from frequent attacks of bronchitis. He denied a venereal history. He used alcohol and tobacco in moderation.

Eight years ago he slipped and fell on the ice, striking his back. He did not lose consciousness, but suffered severely from pain in the region of the thorax on both sides. He remained in bed one week, and then returned to work. For two years previous to his injury he had suffered from tingling and cold paresthesia in the left upper extremity, which he attributed to his habit of sleeping on the left side, resting his head on his hand and thus compressing the left arm. The fall apparently aggravated his symptoms, and subsequently he noticed that his fingers were numb, and that he could not feel objects distinctly with his left hand. With this hand he also failed to distinguish between hot and cold with certainty. The numbness and loss of sensation gradually extended to the left supraspinatus region, the occiput, and more recently to the submaxillary region, the left anterior chest, the left posterior thorax, and finally the right shoulder and arm were involved in the area of cold paresthesia. The patient complained of pain in the left interscapular region during the past eight months, and of a sense of constriction about the left chest, neck and occiput, as if the entire region was encased in plaster. In the neck he had a choking sensation, interfering with swallowing and speaking, and the left ear seemed to be blocked at times. Eight months ago he noticed that the left upper extremity was weak, with wasting in the thenar region. Recently fibrillary twitchings developed in this arm, with excessive perspiration in the left axilla. There was no rash; no joint changes; no trophic skin changes. There was some delay in urination, and the bowels were costive. There was a diminution of sexual potency. No diplopia; the other special senses were normal. The spinal column was scoliotic, with a convexity to the left in the cervical, and to the right in the dorsal region. In walking, the patient dragged the left leg somewhat; no Romberg.

Dr. Abrahamson said that the diagnosis of syringomyelia was easily inferred from the symptoms in this case. It was of special interest because of the fact that the upper border of sensory disturbances corresponded exactly to the sensory distribution of the first cervical segment on one side only, and also because of the very slow progress of the disease, especially in the motor areas.

Some Forms of Toxic Deliria.—Dr. George H. Kirby presented this paper. He stated that in addition to the alcoholic group of psychoses, we found a smaller number of cases in which the mental disorder could be attributed with more or less certainty to some exogenous toxic substance. In the service of the Pathological Institute on Ward's Island a variety of such cases had been met with. These had included intoxications from the use of morphia, cocaine, the bromide salts, chloral, phenacetin, bromo-seltzer, and also from the excessive use of tea and coffee.

Dr. Kirby then reported in detail three cases of toxic deliria. The first of these was due to the excessive and continuous use of phenacetin; the second to the use of chloral taken after alcoholic excesses, and the third to the use of bromides for the relief of insomnia. These cases all had certain symptoms in common. The patients were not clear as to their surroundings; they apprehended readily what was said to them, and were able to grasp, more or less, the simple relations which came under their own eyes, but they invariably mistook the situation in which they were placed. They were especially confused over the identity of persons, while time orientation suffered comparatively little. Consciousness regarding their own personality remained intact, and with one exception, the patient's grasp on the remote past was clear. Perhaps the most striking symptom was the tendency to produce spontaneously detailed accounts of extraordinary occurrences, dream-like in character, and mostly of a terrifying and fearful content. Especially noteworthy was the fact that motor agitation and fear reactions might be entirely absent. Improvement in all the cases was gradual, and the belief in the delirious experiences remained some time after the patients were otherwise clear. The condition had to be differentiated from (1) delirium tremens, (2) general paralysis, (3) Korsakoff's psychosis, (4) the great variety of symptom types in the infective-exhaustive group of psychoses.

Dr. Joseph Collins said the cases Dr. Kirby had related were not unlike some that the speaker was constantly seeing in the general medical wards of the City Hospital. They were cases that had given him a great deal of difficulty in classifying satisfactorily, save on a purely etiological basis, and latterly he had included them under the head of Korsakoff's syndrome. They were cases of sub-acute delirium, not of sufficient severity to cause them to be committed to an asylum, and were characterized by their possession of mistaken identity, with considerable credulity, which varied from time to time, and a lack of orientation as to time and place, which also varied from day to day They did not have the physical signs of a generalized neuritis Most of these cases occurred in women, and recovery took place in from four to sixteen months. Two such cases were under his observation at the present time. They were usually persons who were addicted to the excessive use of alcoholic stimulants, and then took chloral or the bromides or phenacetin or bromo-seltzer. They did not develop the symptoms of multiple neuritis, but there was some tenderness on deep pressure over the nerves. Dr. Collins said he saw no reason for not considering these cases as mild, abortive forms of Korsakoff's syndrome, or the acute delirious condition described by Bonhöfer. They invariably recovered from the first attack. The speaker said he could not recall having seen a case which had not recovered.

Dr. Smith Ely Jelliffe said the question was interesting from a chemical and toxicological standpoint, chloral was an alcohol derivative. The reduction contents of chloral were of the nature of a trichlorethyl-aldehyde, the drug being formed by the action of chlorine on ethyl alcohol. The integrity of the liver must be assumed or assured in order that chloral should be reduced to the product mentioned, because its reduction was dependent on the function of certain undetermined liver activities. Under conditions in which these activities were modified or impaired, it would be interesting to see what the relation would be between the diminished liver reduction and the poisonous effects induced by chloral in these cases.

Dr. Henry S. Noble of Middletown, Conn., said that in the cases of

Korsakoff's disease with which he had come in contact the prognosis had not been as favorable as in those referred to by Dr. Collins. They were seen in a large institution and were possibly far advanced, or there may have been previous attacks, but they were certainly of a chronic character and did not recover. The speaker said he could trace a resemblance between the case reported by Dr. Kirby and Korsakoff's disease; they seemed to depend on a toxic influence, and to that extent, at least, fell in the same category. It was not at all necessary in Korsakoff's disease that the symptoms should be attended with a neuritis, although a neuritis was often present.

Dr. R. A. Defendorf of Middletown, Conn., said he did not think Dr. Kirby had established the fact that alcohol was not the etiologic factor in the cases he had reported, instead of chloral and phenacetin. The speaker said he had seen cases of alcoholic deliria with symptoms very similar to those in two of the cases reported, particularly those in which hallucinations were absent. He did not agree with Dr. Collins that the symptom-complex in those cases closely approached that of Korsakoff's disease, principally on account of the prognosis. He had yet to see a case of Korsakoff's disease where the patient had recovered.

Dr. Kirby, in closing, said he could readily understand that many of these patients with symptoms of toxic deliria did not reach the asylum. In some instances the symptoms were due to a combination of toxic influences. In only one of his cases could the symptoms be attributed to a single drug, and that was phenacetin.. Usually Korsakoff's psychosis, whether accompanied by a polyneuritis or not, was a chronic delirium. In the cases he had reported the disorder was acute, and a study of the delirium disclosed several points of dissimilarity. The attention of these patients was easily engaged, but very hard to hold, while in Korsakoff's delirium the patients had a fairly good power of attention. Dr. Kirby said he had never seen a case of Korsakoff's disease completely recover. In the cases he had reported the patients fabricated along a certain train, while in Korsakoff's disease they were more apt to vary.

PHILADELPHIA NEUROLOGICAL SOCIETY.

April 25, 1905.

The President, Dr. Joseph Sailer, in the Chair.

Two Cases of Korsakoff's Psychosis, One with Necropsy.—These were reported by Dr. R. V. Patterson.

Dr. Pickett stated that Korsakoff's first paper on this subject appeared in a Russian journal in 1887; but the more cosmopolitan announcement of it in German was made in 1889. Dr. Mills had mentioned the coexistence with polyneuritis of mental symptoms which he ascribed at that time to encephalitis before Korsakoff's paper appeared. Korsakoff himself in 1889 said that the peculiar mental disturbance which was the main feature of his disease, the pseudo-reminiscence or fabrication, had been described by many observers before, among them Magnus Huss. As Korsakoff said, and many have said since, this peculiar symptom, combined with other symptoms of confusion, may occur without polyneuritis; and it is not always, as happens in these two cases of Dr. Patterson's, alcoholic in origin, although Osler in his text-book intimates that it is. Korsakoff laid especial stress upon this point, and reported fourteen cases, not alcoholic, in which this mental condition was marked. Dr. Pickett stated that seven cases had come under his observation. One was a case of Dr. Dercum's, two of Dr. Mills', three more were in the insane department at Blockley, and one in private practice. All of these cases were alcoholic, and he thought alcoholism a sufficient cause for the mental state. Korsakoff's fourteen cases not alcoholic occurred in convalescence from typhoid fever, in puerperal fever, tuberculosis, diabetes, etc., and in various exogenous poisonings; that is, in a great variety of conditions in which toxemia is prominent, and he therefore gave the name of toxemic psychic cerebropathy. It appeared to Dr. Pickett that most of the later writers restrict the name Korsakoff's disease to polyneuritic psychoses, and largely to those of alcoholic origin.

Dr. Mills stated that for twenty years he had observed cases of what is known as Korsakoff's disease, in both private practice and at the Philadelphia General Hospital and elsewhere. He thought it true that the peculiar mental symptoms occurring in Korsakoff's disease are also observed in some other affections. He stated that at the Philadelphia General Hospital, a few months ago, there was a patient in whom the diagnosis of syphilis of the nervous system had been made in whom the distinctive mental symptoms of Korsakoff's disease were present, especially the memorial fabrications and general mental confusion.

Dr. Dercum thought it unfortunate that a special name has been given to this symptom group. He considered it a toxic confusional insanity, not differing from other forms of confusional insanity. As regards the association of polyneuritis, he has at present a patient in whom fabrication is going on constantly, at times expansive, at times depressive, but with no trace of multiple neuritis. We see this symptom group in toxic psychoses other than those due to alcohol, and these patients have, as did the one just mentioned, hallucinations of hearing, marked confusion, and their fabrication is to be translated into the terms of a delirium, mild in character presents nothing that is distinctive or characteristic.

Dr. Gordon stated that he had seen four cases of this syndrome, two being men and two women. The men both had alcoholic histories, and the women did not. One of the women developed this syndrome after a protracted diarrhea, and the other had a history of pneumonia, but afterwards developed wrist drop, foot drop and tenderness of nerve trunks; the mental symptoms were a confusional state and illusions of identity. Korsakoff originally described this condition as due to alcohol, but other observers have reported cases where alcoholism was not present. In Dr. Gordon's opinion the symptom group of the affection may follow toxic conditions other than alcohol.

Dr. Spiller stated he had had an extraordinary experience with alcoholic multiple neuritis this year at the Philadelphia General Hospital and elsewhere. There had been eight or ten cases, three with autopsies. In several cases there was the symptom group of Korsakoff's psychosis.

Dr. Dercum thought that, during delirium, hallucinations of sight are quite common, but that these pass off as the delirium subsides. He thought it quite true that in the milder forms of alcoholic disease hallucinations of hearing predominate over hallucinations of sight.

Dr. Hawke stated that while many cases of acute alcoholic confusional insanity come to the Philadelphia General Hospital, they had very few cases of multiple neuritis. He believed that the symptom-complex delirium is often mixed up with confusion. He thought that in these cases of Korsakoff's disease there is no delirium, but confusion. In the cases of confusion that he had seen very few had hallucinations that could be depended upon. All the cases that had been observed in the insane department had shown symptoms of illusion of identity with fabrication.

Dr. Patterson stated that the improvement in the mental symptoms of his case was much more rapid than the improvement in the physical condition. In five or six weeks the mental symptoms had cleared up markedly, but the physical symptoms are still quite pronounced.

A Case of Amyotrophic Lateral Sclerosis Associated with Long-Stand-

ing Poliomyelitis.—This was presented by Dr. Klaer from Dr. Spiller's service at the University Hospital.

Dr. Gordon stated that in 1902 he exhibited a case of amyotrophic lateral sclerosis in a boy of twelve, who at the age of three had an attack of acute poliomyelitis. He thought that cases of amyotrophic lateral sclerosis were rare, and that the one exhibited from Dr. Spiller's service was interesting as showing that an old myelitic focus might at any time under the influence of trauma or other cause develop into something else.

Dr. Mills believed that the unilateral form of the amyotrophic lateral sclerosis is very rare, but stated that he had recently seen a case of unilateral amyotrophic lateral sclerosis in a boy of about fourteen.

A Case with Unilateral Exophthalmos and Extensive Involvement of the Cranial Nerves.—This was exhibited by Dr. Wm. Zentmayer and Dr. T. H. Weisenburg. Dr. Weisenburg remarked upon this case as follows: The woman when fourteen years of age had the first symptoms of menstruation, and coincident with these she had exophthalmos. She then did not menstruate regularly for several years, but since eighteen years of age has menstruated regularly. Every time she has her menses the eyes bulge. She married at twenty-four, became pregnant four years later, and then noticed that her eye was full for two months. After this pregnancy her left eye was completely closed. The distribution of the right third, fourth and sixth nerves and the sensory part of the fifth are now affected. She has complete anesthesia of the sensory division of the right fifth nerve. There is a little weakness of the seventh nerve of the right side. The eighth nerve is normal. The right ninth and tenth nerves are involved. The right twelfth nerve is involved, as shown by fibrillary tremor and atrophy of the tongue. The diagnosis first made in this case was sinus thrombosis in the cavernous sinus, and this condition would account for the involvement of the second, third and fourth nerves. The fifth, sixth, seventh, ninth, tenth and twelfth nerves could be accounted for by extension of the thrombosis backward. The only other diagnosis in this case would be neuritis, but a neuritis which would involve the cranial nerves only on one side would be extraordinary. Another diagnosis that might be made would be meningitis, but this also would be bilateral.

Dr. Lloyd stated that this case reminded him of two cases he had had in his service in the Philadelphia General Hospital, both of which he had put on record as unilateral ophthalmoplegia, external and internal, associated with involvement of the fifth nerve. One case was reported in the *University Medical Magazine,* and the other in the JOURNAL OF NERVOUS AND MENTAL DISEASE. In one case an autopsy was obtained. In both cases there was involvement of the fifth nerve, in one case there was pain and in the other anesthesia.

Dr. Spiller had examined this case and did not think any diagnosis possible but sinus thrombosis.. The nerves nearest the cavernous sinus were the most affected, while those farthest away, where the pressure was less, were less affected.

Dr. Alfred Gordon presented a Case of Exophthalmic Goitre with Involvement of the Cranial Nerves.

Dr. Thomas J. Orbison read a paper entitled: Delusional Insanity of the Persecutory Type with Clinical Relapses.

Dr. Charles S. Potts and Dr. Wm. G. Spiller read a paper entitled: Pseudo-sclerosis (Diffuse Sclerosis) with the Report of a Case with Necropsy.

Dr. Charles W. Burr and Dr. C. D. Camp read a paper entitled: The Causes of Triplegia.

Dr. J. H. W. Rhein reported a case of Syphilitic Encephalitis and Double Hemiplegia with Necropsy.

Dr. Mills made some remarks upon New Terminology in Nervous Diseases.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

May 18, 1905.

The President, Dr. Morton Prince, in the Chair.

A Case of Brain Abscess of Obscure Origin in a Man of Forty-three.—This was reported by Dr. H. W. Mitchell. Previous history was negative. No aural, pulmonary or other source of infection was discovered. During three days forgetfulness and severe paroxysmal headache, with vomiting; temperature 100 degrees; marked hebetude and photophobia, but patient clear mentally. Pupils equal and normal; reflexes lively and strength of extremities unimpaired. At the end of one week there was paresis of the right arm and slight enlargement of the right pupil with motor aphasia. On the tenth day the right leg was also involved with Babinski. The left optic disc was poorly defined, with vessels enlarged. Coma and convulsions supervened and death occurred at the end of the tenth week The leucocyte count was 12,600 to 13,200. Temperature 99 to 101 degrees.

Autopsy by Dr. Barrett showed a pus cavity, subcortical, in the left frontal lobe, 5.5 cm. bv 3.25 cm. Internal capsule not involved. Gram-staining coccus and a long, thick bacillus were found, but no growth on the blood serum.

A Case of Adiposis Dolorosa (Dercum's Disease).—Dr. E. W. Taylor reported this case occurring in a patient who entered the Long Island Hospital with an acute alcoholic neuritis with mental symptoms. She was then a woman of ordinary size, weighing perhaps 150 pounds. After convalescing from the neuritis she developed a tendency to adiposity, which has continued so that now she weighs a few pounds short of 300. The fat presents the characteristic lobulations, with much pain, varying somewhat from day to day. (Photographs of the case were shown.)

Dr. E. W. Taylor also showed photographs illustrating certain pathological alterations in a case of metastatic carcinoma of the vertebræ. The cord showed certain relatively slight secondary nerve root degenerations in striking contrast to the marked degeneration of the cells of the ventral horns in the lumbar region. These cells almost without exception showed extreme degeneration of the axonal type—central chromatolysis with dislocation of the nuclei. Cells in other parts of the nervous system were not affected in anything like the same degree, hence the supposition of a general toxemia seemed less tenable than local interference with the ventral roots of the lumbar region.

Dr. Putnam said that as Dercum's disease is rare, he would like to put on record the fact of having recently seen a typical case in consultation. The symptoms were very characteristic, but the fat development was not as extreme as in the cases reported by Dr. Taylor.

Synopsis of Thirteen Cases of Multiple Neuritis.—These cases were seen at the Massachusetts State Hospital, Tewksbury, Mass, February-May, 1905. Dr. Dutton said the etiology of these cases is very obscure. All were males in the asylum department and came from two wards. Some had been working on the farm. Conditions of patients were dissimilar, ruling out exposure and chronic poisoning, as by arsenic from wall paints, etc. They had had the same diet as patients in other wards where there were no cases, which appears to satisfactorily rule out poisoning by either water or food. This leaves very little except some bacteritic infection. There were no lung symptoms in any case, and repeated attempts to demonstrate influenza were futile. A lumbar puncture in one case gave an absolutely clear, pale serum, sterile on culture and inoculation and having no sediment. Blood examination was not remarkable in any case.

The onset was with edema of the lower limbs, coming on suddenly in most cases and extending over the whole body, involving especially the neck, where it was persistent after the disappearance in other portions of the body. No cardiac lesion had previously been detected in any case, but cases then showed cardiac murmurs, usually mitral, with slight enlargement to the left. The pulse was rapid in two cases, slow in two, and in the balance was not appreciably altered. There was no temperature over 100, and most of the cases showed no elevation of temperature. Respiration did not appear to be affected. There was no pleural or pericardial effusion detected.

With the disappearance of the edema, in from seven to fourteen days, tenderness of varying extent, especially of the lower limbs, became apparent, with muscular atrophy and paralysis of varying degree following. Wrist drop was present in six cases, with toe drop present in four of these six and in two others. Electrical reactions of degeneration in all cases. Patellar reflexes lost in all cases; pupil reflexes were not affected.

Four cases showed gastro-enteric symptoms, with dejections of bloody mucus and undigested food, etc. Three of these cases had hiccough and died. Urine examination in no case showed anything pathological. The muscle of six cases was examined for trichinæ, but none were found.

Death occurred in six cases. Seven cases are still under observation. In these cases the cardiac murmurs have nearly disappeared. The tenderness is still marked, and toe drop of varying degree is present in four cases. There is marked muscular atrophy in all but one case. All of these cases appear to be slowly regaining their strength.

An autopsy on one case showed nothing remarkable macroscopically except numerous small, somewhat irregular, depressed ulcers, with overhanging edges, apparently healed or nearly so, scattered through the large intestine most numerous in the lower portion.

Dr. Putnam said that he had had an opportunity of seeing these interesting cases, but could throw no light on their etiology. The possibility of beri-beri had, of course, suggested itself, but there was nothing except the symptoms to warrant that diagnosis. He referred to the fact that some years ago he had reported a small epidemic which had broken out in some fishing vessels on our shores, and said that he also saw several cases last summer at St. Louis, where an epidemic occurred among the Philippinos visiting the exhibition. Some of these patients fell ill after having been for some time away from their native home. Dr. Bliss of St. Louis had also called attention to the fact that epidemics similar to the one reported by Dr. Dutton had occurred in several state or city institutions.

Dr. Edes said that these cases reminded him strongly of some which entered the Boston City Hospital in 1884. The men had been sailors of the British ship "Earl Granville," which sailed from Ilo-ilo around the Cape of Good Hope. The food is said to have been good and abundant and antiscorbutics were furnished, but perhaps not very abundantly. The water was most of the time good, but for a long time they used rain water which dripped from the rigging and a newly-painted deck-house. Out of eighteen persons on board six were sick; three came to the City Hospital, and one died there. One died on board. The three hospital cases were reported at some length in the *Boston Medical and Surgical Journal*, Vol. CX., p. 607, as cases of an unusual form of scurvy, for want of a better diagnosis, although the most characteristic symptoms of that disease, such as hemorrhages and swelling of the gums, were absent or very slight. The principal symptoms were severe pains and swelling of the legs, with dyspnea and cough. In the case which died there was edema of the lungs and bronchopneumonia. The nervous centers were not examined. In only one case were the symptoms on the part of the nervous system noted with sufficient distinctness to make a neuritis highly probable. In this case (which was not the fatal one) there was stiffness and pain in the legs, with difficulty in using them. The muscles were well developed and hard, perhaps unusually so, and reacted only feebly to a strong faradic current. There was no patellar reflex. There were well-marked signs of nephritis in two cases and perhaps in the third. On reviewing these cases later it seemed evident that they were not scurvy, but beri-beri, a disease of which but little was known here at that time, and the cause of which if, as is probable, partly at least dietetic, is certainly not the same as that of scurvy.

Dr. Webber said that the epidemic of multiple neuritis just reported resembles the one to which Dr. Edes referred, which occurred about twenty years ago in this city. A large number of cases were admitted to the Boston City Hospital. The earlier cases were under Dr. Denny's care, and Dr. Webber followed him and saw a dozen or more cases. Many had their limbs swollen and the diagnosis of rheumatism was made before admission. The symptoms were very similar to those just mentioned. There were two deaths. Dr. Webber made one autopsy and later reported the cases. There was a suspicion or a suggestion at that time that the cases were beri-beri. He did not investigate the residence of the patints to learn if they came from the same quarter of the city.

Dr. Knapp said that the edema and cardiac complications in this interesting series of cases associated with other symptoms make the diagnosis of beri-beri very probable. Some years ago Boudourant described an epidemic of beri-beri at the asylum of Tuscaloosa, Alabama, and not long after quite a serious epidemic was reported in an asylum near Dublin. A few years ago a number of cases of beri-beri were brought to the City Hospital from a vessel that had arrived from some East Indian port. Dr. Knapp saw nearly all the cases of multiple neuritis at the City Hospital in 1883, at the time when we first began to recognize the disease there. Since that time the cases have been fully as frequent, so that few of them could be regarded as of an infectious type. Many, in fact, at that time were alcoholic and presented the typical alcoholic polyneuritic psychosis long before Korsakow described it. Gerrard, who observed many cases of beri-beri in the Selangor prison and Kwala Lampor jail in the Federated Malay States, found that the disease became endemic in certain dark cells, and that monkeys placed in these cells developed the disease. He regards it as absolutely disproven that the disease is due to any form of diet, either fish or rice, and found some facts indicating that it was of bacterial origin.

A Section of an Endothelioma in the Rolandic Region.—This was shown by Dr. Knapp. The patient, a powerful negro, aged forty-six, came to the City Hospital in March, 1903. The family and previous histories were unimportant. Eight months before, he began to have numbness in the right little finger, which in three weeks had involved the ring and the ulnar half of the middle finger. A month later he suddenly lost power in the right arm for ten minutes. After that the numbness disappeared. A month later, while in bed, he found the right arm numb and powerless for ten minutes on waking. After that time he had attacks of formication in the right hand, and the arm felt powerless. In November, 1902, he began to have attacks of clonic spasm in the right fingers and thumb, later involving the whole hand. During an attack of pneumonia the attacks ceased, but recurred after recovery. Six days before coming to the hospital he had an attack with clonic spasm of the whole arm and thickness of speech. He never lost consciousness in these attacks. He had at times slight vertigo, and occasional headaches of moderate severity on the left side of the head. He had had specific treatment for two months without relief.

Some facial asymmetry had been noticed for four months, the right naso-labial fold being more marked. There was some arteriosclerosis. The pupils reacted through a narrow arc. The right arm was weaker. The fundus of the eye was normal. The urine was negative. There were the traces of an old pleurisy. Sensation was about normal. The reflexes were not remarkable.

Operation was advised and refused. He came only occasionally to the hospital, and had evidently been impressed with the fact that he was an interesting case, and accordingly demanded to be treated with great deference.

In August the right leg had grown weak and he dragged the right foot. The numbness had increased and he was unable to button his clothes with the right hand.

In December a large trephine opening was made over the Rolandic fissure at the Homeopathic Hospital, but the growth was found to be inoperable on account of hemorrhage. A portion of the growth was removed and found to be endothelioma.

In January, 1904, the attacks were recurring several times a day, and he had some headache. In March he had another attack of pneumonia. On the third of August, 1904, he was having frequent attacks, beginning with a feeling of pins and needles in his right middle and ring fingers, extending to the forefinger, thumb and whole hand. Then followed flexor movements of all the fingers. The attacks at times involve the wrist and arm. The right face is sometimes drawn after an attack, and the speech may be affected for an hour. He knows what he wants to say, but cannot say it.

Three weeks before, he had similar paresthesia in all the fingers of the left hand. The left leg swells at times and has painful paresthesia. He had grown quite obese and had right hemiplegia. The arm was more affected than the leg, and the leg more than the face. All the fingers of the right hand were in flexor contracture. The right knee-jerk was greater, but there was no clonus.

August 17th he reported severe headache for ten days. On the 31st of August he could not walk or speak as well, and could see hardly anything. There was a feeling like a red-hot iron in the left hand. The headache persisted. There was a well-marked hernia cerebri, which pulsated. The neck had a porky, edematous look. His wife reports swelling of the legs. Some time after this mental symptoms developed, and he was committed on the 31st of October, 1904, to the Boston Insane Hospital with hallucinations of sight and hearing, delusions of poisoning and of wealth. He believed his wife responsible for the poison and had attacked her. On admission he was somewhat aphasic, much confused and had marked impairment of memory. He was contented and happy. He had delusions that his right hand had been cut off, that his money had been taken from him, that he had been abused, dragged upstairs, etc. When reciting these trials he would weep. He had hallucinations of hearing, much headache and marked failure of vision, distinguishing only light from darkness. He grew duller, then brighter, and early in January was permitted to return home. There he grew duller; the hemiplegia and blindness increased; he eventually became comatose and died with symptoms of marked increase of intracranial pressure on May 1, 1905. It is a curious feature of the case that after a large trephine hole was made in the skull 3 by 4 cm. in diameter, the headache should have grown worse, the optic neuritis, which was not visible nine months before, should have developed so as to produce complete blindness, and mental symptoms of so pronounced a character should have developed. Out of 104 cases of brain tumor with atrophy which Dr. Knapp had recently tabulated, but one other patient was obliged to go to an insane hospital, and but one other had delusions of a persecutory type, although some form of mental disturbance was the rule. The tumor could probably have been easily removed in the early stages, with a fair chance of recovery.

Dr. Shirres said that three years ago, and within a period of three months, two very interesting cases of cerebral tumor came under his care at the Royal Victoria Hospital. In both was there a history of trauma. The first patient was a boy of ten years, who fell from a height. He was unconscious for a few minutes, but otherwise did not seem to suffer from any bad effect. Six months afterward he developed epilepsy, and was treated for what was supposed to be idiopathic epilepsy.

At the end of that period he was not seen for about six months, when he again came to the clinic, having been sent by the ophthalmologist, Dr. Buller, under whose care he had come, complaining of loss of vision. On examination choked disc was detected.

A careful examination revealed a slight paretic condition of the left hand, with anesthesia confined to that region also. He was taken into the hospital for observation. Some six weeks later, while playing in the grounds, he had an epileptic seizure and died.. At the autopsy a large endothelial tumor, 4 in. by 3 in., was found in the right cerebral hemisphere. On the internal surface of the right parietal bone was found a bony exostosis about the size of the terminal phalanx of the little finger. This pressed down onto the dura mater and brain substance. When the bone was removed the surface of the dura mater could be seen indented. This latter membrane for three-quarters of an inch in circumference from this indentation was markedly thickened and adherent to the tumor substance below. The tumor was a non-infiltrating endothelial growth. Under the microscope one of the most typical pictures of this growth could be seen.

The other case occurred in a woman, aged thirty-five. A history of epilepsy following an accident was present. Two years later she died, and at the autopsy a tumor of the same nature and with bony exostosis from the under surface of the parietal bone was found. In fact, it was nearly impossible to tell one brain from the other after they were hardened. One was hardened in formalin, the other in Müller. It was due only to this that one could say which was which, they were so much alike..

The above two cases go to show the etilogical factor in the production of endothelial growths. The bony exostosis produced an irritation of the endothelial cells in the subdural and pial spaces, which led on to hyperplasia or tumor formation.

Eye-strain; Its Importance and Its Limitations.—This was read by Dr. Walton. His conclusions were:

1. Among individuals totally blind since infancy 66 per cent. were free from tendencv to headache, as contrasted with 31 per cent. of those having sight, and 29 per cent. of those with partial or with acquired blindness.

2. If these figures should prove constant, the inference would seem justifiable that half the headaches in health are due to eye-strain.

3. The headache, when present among those totally blind since infancy, partook sufficiently often of the migranoid character to preclude the supposition that *all* migraine is due to eye-strain.

4. The results of this study would indicate that while migraine, and migranoid headaches, have a constitutional basis, and while other factors than eye-strain may act as exciting causes, still, eye-strain is one of the most, if not the most, important of these exciting causes, and steps for its relief are imperative.

5. In no case has correction of refraction been given a thorough trial until (*a*) the glasses are properly centered, (*b*) their continued readjustment is practised, (*c*) the patient looks as much as possible through their centers instead of from side to side, (*d*) efforts are avoided at straining the eyes to see distant objects with the glasses, (*e*) spectacles instead of eyeglasses are used, and (*f*) the use of spectacles is constant, not intermittent.

6. The constitutional headache of the deviate is probably allied to the headache of "brain fag," but is out of all proportion to the sources of fag. Little can here be expected of spectacles

7. In the proportion in which obsessive tendencies and other signs of constitutional peculiarity accompany errors of refraction, efforts at the correction of refraction will prove unavailing for the relief of nervous symptoms.

Dr. Edwin E. Jack said that it is difficult or impossible in some cases to tell beforehand whether glasses will relieve or not. There is no doubt about some of the cases of "constitutional headache" which Dr. Walton speaks of, but Dr. Jack could not always distinguish between these and others. They are mixed up with the neurasthenic cases, some of which do and some do not suffer from eye-strain. Another point mentioned by Dr. Walton is the fact that the first pair of glasses may not relieve even in favorable cases. There are many things which make the term "fitting glasses" improper. As exact a result as possible must always be got by careful examination, but this result is often only a basis on which to work; it has to be modified to meet individual requirements.

Dr. Kilburn heartily endorsed all that Dr. Walton had said. He was especially glad that Dr. Walton had placed so much emphasis upon the importance of having spectacles accurately centered and aligned. This is half the battle. Unless the optical centers of the lenses are exactly opposite the pupillary centers the patient gets the effect of prisms base out or in. Also, unless a line drawn through the optical centers of the lenses is parallel to a line drawn through the pupils the patient, if astigmatic, gets the effect of incorrect astigmatic axis; or, if merely plain hypermetropia or myopia be present, the patient gets the effect of prisms base upwards or downwards. He felt that it is not going too far to say that lenses which are not of exactly the proper strength will, if correctly placed, give more relief to a patient suffering from eye-strain than will correct lenses, improperly placed, provided that, if the patient be astigmatic, the axes are correct. As regards nasal reflexes, he said that he had had a large number of cases in which, in his opinion, nasal reflex played a prominent part. He believed without question that in a large proportion of his cases of heterophoria pressure on the nasal nerve fibers has been the cause of the heterophoria. This conclusion is borne out by the results in a large number of cases. Also, in all cases in which the refractive error changes frequently, at short intervals he was strongly of the opinion that there is probably some reflex from the nares.

Dr. Alex. Quackenboss said he had listened to Dr. Walton's paper with a great deal of interest, and it seemed to him that the careful study of a patient's general condition, symptoms and temperament is of the greatest aid in distinguishing between those cases that can and those that cannot be relieved by glasses. Another point to consider is the character of the headache: whether it occurs in the morning and passes off during the day, or whether it comes on during the latter part of the day, especially after use of eyes, or whether one is awakened during the night by it. Setting aside the extravagant claims of certain writers in which all cases of this class are cured by the use of glasses, and the statement of others that they have seen no benefit derived from glasses, the fact remains that a great many obtain relief by a proper correction of their refractive error.

Dr. Clap regarded centering and alignment of frames as of as much importance as correct lenses. Often a return of symptoms will be found in a patient without change in the refractive condition. As illustrating the small refractive changes sometimes of importance, he mentioned a medical man whose astigmatic glass (originally fitted by Dr. Clap) he reduced in strength ¼ diopter four weeks ago. The man was having scintillating scotoma every day, followed by headache. The change was followed by absolute disappearance of scotoma and headache, although he is working harder than ever.

Dr. F. L. Jack said that Dr. Walton referred briefly to nose deformities as a cause of headache. This important factor has long been recognized, and there is little that is new to be said about it. Obstructions in the nose, either from deviated septum, spurs, polypi or enlarged turbinate, are by pressure undoubtedly often the cause of pain in the head.

Dr. Loring agreed with Dr. Walton's paper, and in what he had said of the the relief to be expected in the cases of "migranoid" headache from the correction of eyestrain. The majority of cases of headache that consult the oculist are entirely relieved. In regard to cases of true migraine, however, his experience is less favorable. In typical cases with scintillating scotoma, hemianopsia and pain on one side, Dr. Loring had found that correction of the refractive error has given in every case more or less relief, but never a complete cure.

Dr. Putnam said that Dr. Walton's paper seemed to him, in the main, excellent, although in some respects rather summary. He had himself been disappointed in not securing more relief for patients through the correction of refractive errors, but presumed that this was due to the fact of their belonging to the class characterized by Dr.. Walton as "constitutional." The term "algesic," used by Dr. Prince some years ago with reference to persons who suffered pain with special readiness, deserves to be recalled in this connection. It is not quite clear why the headaches cured by glasses should be spoken of necessarily as migranoid, since they are certainly not all of the same type, and since, also, severe cases of typical migraine are rarely amenable to treatment of this sort. As regards nasal treatment, Dr. Putnam thought that it was certainly occasionally of value, if only as an adjunct of the correction of refraction.

Dr. Shirres asked Dr. Walton whether he thinks he can compare those blind patients who are in institutions to individuals who are following the busy, active pursuits of daily life. The surroundings of one cannot, in his opinion, be compared to the other. In these modern days the stress and artificial life is so liable to produce bad results that one can easily account for the more prevailing tendency to headache or eyestrain in those latter classes.

Dr. Knapp had not found typical migraine to be nearly so common as Dr. Walton's statistics indicated, and he agreed with Dr. Loring that eye-strain was a less important factor in the production of true migraine. At the present time the public seemed to be thoroughly instructed as to the possibility that headache might be due to eye-strain, and it was rare to see a patient with any form of headache who had not consulted some ophthalmologist already. In all cases really due to eye-strain he had found that there were usually symptoms pointing directly to the eyes. The headaches were apt to be frontal, and the use of the eyes for near work, especially under unfavorable conditions, induced or aggravated the headache.

Dr. Walton, in closing, spoke of the identical diet and surroundings of the two classes of blind individuals examined at the Perkins Institute, which seemed largely to limit the difference in frequency of headaches to the difference in ocular conditions. He hoped that the speakers who had failed to benefit migraine by glasses had carefully followed the details he had mentioned.. He was not sceptical regarding the production of headache by abnormal nasal conditions, but had no faith in either nasal or ocular treatment for the constitutional headache and allied symptoms characterizing the ideo-obsessive.

Periscope

American Journal of Insanity

(Vol. 61, No. 4.)

1. Presidential Address. A. E. MacDonald.
2. General Paresis and Tabes Dorsalis. Henry A. Cotton.
3. Notes on a Visit to Some Foreign Hospitals for the Insane. Edward N. Brush.
4. Functional Insanity. Robert Jones.
5. Comparative Measurements of the Hard Palate. Walter Channing and Clark Wilson.

1. *Presidential Address.*—An address delivered at the 1904 meeting of the American Psychological Association. Retrospective, with some suggestions as to improvement of methods in the recording of admissions and discharges of insane patients.

2. *General Paresis and Tabes Dorsalis.*—A review of the literature of the subject, with a report of the results of the clinical and anatomical study of twelve cases presenting a combination of tabetic and paretic symptoms. The author comes to the following conclusions: 1. Clinically, tabes and general paresis present many analogies in etiology, symptomatology and course. 2. Their occurrence in the same individual is more than a coincidence. 3. In these cases of tabo-paralysis the symptoms presented are identical with the symptoms of general paresis and tabes when seen apart, only differing in degree according to the extent of the anatomical lesion. 4. The clinical symptoms of tabo-paralysis have the same anatomical basis as in the separate diseases. 5. Anatomically the affection of the posterior columns of the cord as seen in tabo-paralysis does not differ from the picture presented in pure tabes. The same systems are affected and the segmental character of the process is the same; also, the process in the cortex is identical with that of general paralysis. 6. While the above facts show the intimate relations between general paralysis and tabes dorsalis, the unsettled status of their pathogenesis at present prevents their identity being absolutely established on an anatomical basis.

3. *Notes on a Visit to Some Foreign Hospitals for the Insane.*—An account of a recent visit to some foreign asylums, mainly the German Psychiatric Clinics, with comparison of their arrangements and methods with those existing in Great Britain and in the United States.

4. *Functional Insanity.*—A discussion of the relations of insanity with the neuroses, especially with hysteria, hypochondria and epilepsy, with remarks upon the possible functional basis of acute mental disturbances. The author finds a close relationship between hysteria and mania in women, and between hypochondria and melancholia in men, and thinks that the great number of women admitted to asylums during the adolescent period of life are suffering from temporary mental disturbances. From all such considerations he feels justified in emphasizing the importance of early and careful treatment of those showing abnormal mental symptoms of any sort, since long-continued functional troubles may at length give rise to organic changes.

5. *Comparative Measurements of the Hard Palate.*—A preliminary report of the results obtained by measuring accurately made plaster casts of the palate in 614 normal and 1,010 abnormal individuals. The figures obtained are placed in tabular form. Although in the abnormal these figures were subject to more variation, the authors found otherwise no marked differences between the measurements in normal and in abnormal persons. Allen (Trenton).

Archives de Neurologie

(July, 1905, No. 115.)

1. Family Morphinism by Contagion. Briand and Tissot.
2. The Fear of Observation (Regard). Bechterew.
3. On the Medical Personnel of Public Insane Asylums. L. Lagriffe.

1. *Family Morphinism.*—The authors relate an instance in which a mother, son and daughter successively became morphinomaniacs on account of association with an habitue in the person of an adopted son. The adopted son developed the habit from taking morprine and cocaine injections for neuralgia, on a physician's prescription. Toothache and sleeplessness were the occasions of introducing the drug in the family. All of the members except a son became confirmed in the addiction, and wandered about from one place to another getting the drugs by various pretexts, until they finally wound up in an institution. In spite of taking large doses for five years, none of the party had delusions or hallucinations; and the authors believe that this fact shows an originally good mental constitution, and is no argument against the current theory of irresponsibility in such cases on account of hereditary taint. The responsibility of the physician and pharmacist is also strongly emphasized.

2. *Fear of Observation.*—Bechterew claims priority over Hartenburg in the description of the fear of observation, because he recorded three cases in 1900. Since then he has observed a number of cases, and he now gives a description of four of these. The disease is one of degeneracy, and th most potent cause is given as masturbation. The condition may exist in a pure form, or may be combined with fear of blushing, difficulty of micturition or enforced laughter. A general treatment of baths, bromides and heart tonics is recommended, together with hypnotism.

3. *Medical Personnel of Insane Asylums.*—The author states that the growing restlessness among the medical staffs of asylums is largely due to the fact that these institutoins are still administered under laws dating from 1839 and 1857. He reviews the situation, and concludes that the physician-in-chief should be relieved from the necessity of seeing all the patients every day, and that the assistant physician should have more authority, especially in regard to signing routine papers, etc .

H. J. Nichols.

Review of Neurology and Psychiatry

(Vol. 3, 1905, No. 3, March.)

1. The Prognosis of Disseminated Sclerosis. Byrom Bramwell.
2. Some Aspects of Alcoholism (Concluded). A. Hill Buchan.
3. A Note on Nervous Lesions Produced Mechanically by Atheromatous Arteries. G. Elliot Smith.

1. *Disseminated Sclerosis.*—Bramwell calls attention to the extremely unfavorable prognosis of disseminated sclerosis, as sooner or later the great majority of cases pursue a progressive course from bad to worse. In some instances, however, the downward progress is interrupted by periods of improvement or complete remission of the symptoms, which lead one to give a favorable prognosis. The symptoms then return with greater intensity and death follows early. In only very rare instances is the improvement lasting, but nevertheless the author doubts whether one is justified in saying that a cure never takes place, as in rare cases the symptoms completely disappear, and one ventures to hope that permanent arrest or cure has taken place. He cites several cases to illustrate his views in regard to these apparent recoveries. Many authorities believe, the author adds, that the patches of sclerosis which are the pathological substratum of disseminated sclerosis are due to the irritation produced by some form of toxin carried to and distributed through the

nervous tissues by the blood-vessels. The recurrence from time to time of the symptoms, after periods of improvement and remission, is very suggestive of repeated intoxications. These toxins are probably produced within the body, so it is not unreasonable to suppose that in some cases the development of the toxins from within or the absorption of the toxins from without may cease and that a permanent arrest and cure may occur.

2. *Some Aspects of Alcoholism* (*Concluded*).—Concluding his article on alcoholism, the author gives the following treatment: In nearly all cases the usual warm tub-bath was given on admsision, and chloral hydrate gr. 20, and bromide of soda gr. 30, were then administered four-hourly until sleep was attained. No bad effects were observed to follow their use. They did not appear to set up gastro-intestinal disturbance and were well retained. In some cases where motor excitement was especially marked, hypodermic injections of hyoscine were found to be of value. Alcohol was not given in the uncomplicated cases. The author finishes his paper by describing briefly cases of alcoholic insanity of a more acute form, and states that a sharp dividing line cannot be drawn between this disease and delirium tremens, and some other conditions arising in the course of chronic alcoholism. In the more typical cases the onset of the disease may be similar to that of delirium tremens, but the hallucinations are not so varied and changeful, while those of an auditory character are more frequently in evidence, and the true delusional element is more systematized than in delirium tremens. The loss of orientation is less; fear, melancholy and ideas of persecution are more marked and more difficult to dispell, the risk of suicidal attempt being greater. The duration of the disease varies from a few days to several weeks or months, recovery being more gradual than in delirium tremens; or it may pass into a more chronic form.

3. *A Note on Nervous Lesions Produced Mechanically by Atheromatous Arteries.*—Smith claims that sufficient attention has not been paid to the rôle played by diseased arteries in the mechanical production of lesions. He describes the brain, in which there was complete atrophy of the left and a partial atrophy of the right optic nerves, caused by pressure of enlarged atheromatous internal carotids in the neighborhood of the anterior clinoid processes. In another case atheromatous disease of the upper part of the vertebral arteries and the lower part of the basilar artery compressed and distorted the medulla oblongata, and probably caused death. ALFRED GLASCOCK (Washington, D. C.).

Book Reviews

Health and Disease in Relation to Marriage. A manual contributed to by Privatdozent Dr. med. G. Abeldorff, Privatdozent Dr. med. L. Blumerich, Privatdozent Dr. phil. R. Eberstadt, Geh. Med.-Rat. Prof. Dr. A. Eulenburg, Geh. Med.-Rat. Prof. C. A. Ewald, Geh. Med.-Rat. Prof. Dr. P. Fürbringer, Hofrat Prof. Dr. med. M. Gruber, Dr. med. Havelburg, Geh. Med.-Rat. Prof. A. Hoffa, Prof. Dr. med. et phil. R. Kossmann, Gen. Med.-Rat. Prof. Dr. F. Kraus, Dr. med. R Ledermann, Med.-Rat. Dr. A. Leppmann, Geh. Med.-Rat. Prof. Dr. E. v. Leyden, Prof. Dr. med. E. Mendel, Dr. med. A. Moll, Geh. Med.-Rat. Prof Dr. A. Neisser, Geh. Med.-Rat. Prof. Dr. J. Orth, Dr. med. S. Placzek, Prof. med. et phil. C. Posner, Privatdozent Dr. med. F. F. Richter, Prof. Dr. med. H. Rosin, Dr. med. W. Wolff. Edited by Geh. Medizinalrat Prof. Dr. H. Senator and Dr. med. S. Kaminer. Translated by J. Dulberg, M.D., of Manchester, England. Volume II. Rebman Co., New York and London.

The list of eminent names given as contributing to this manual is a guarantee that the subjects discussed will be treated with abundant scientific knowledge, sanity and commonsense. Naturally. considerable space is devoted to the consideration of gonorrheal diseases and syphilis, as related to the married state, and also to diseases of the genito-urinary organs in both sexes. Scarcely less intimately related to the matter in question are nervous and mental diseases, treated of by Prof. Eulenburg and Prof. Mendel; and a long chapter on alcoholism and morphinism is contributed by A. and F. Leppmann. Diseases of the skin, diseases of the eye, with especial regard to their hereditary aspects, diseases of the organs of locomotion, and occupational injuries are each accorded a chapter. Sexual perversions form the topic of still another chapter, and some pages are given to the consideration of medico-professional secrecy, from a standpoint, however, so distinctly German in both its legal and its social aspect that although interesting, it loses much of its value for the English or American reader. Throughout the volume the attitude of the physician is frequently touched upon, and suggestions made in accordance with his obligations to his profession and to his patients. The book closes with a chapter on the importance of sanitary conditions, which necessarily brings in social, economic and political questions. It is interesting to note the authors' opinion as to the much-discussed question of health certificates for those about to marry. They are on record as being unanimously in favor of it, but not, of course, implying any legal restrictions on marriage. The provisions they advocate would include merely the obligation on the part of the contracting parties to furnish each other with a copy of the certificate, the matter of marriage, after such a certificate was delivered, being left entirely to their own option.

Im Grenzlande, Aufsätze über Sachen des Glaubens. Von P. J. Möbius. Johann Ambrosius Barth, Leipzig. Hoeber, New York. 3 mark.

In this the last of Dr. Möbius' aufgewohlte werke, the author confesses very naively that in his youth he had aspirations to be a philosopher, but that the lack of money prevented him from devoting his time to philosophical thoughts.

We do not know whether we might have lost a philosopher, but we certainly have gained an interesting and entertaining neurologist, who here deals with general philosophical problems in a manner rarely vouchsafed the philosopher by occupation.

Dr. Möbius has here collected six essays written from 1891 to the present time: On Three Ways of Thinking, 1891; Three Discourses Concerning Religion, 1896; Concerning the Elevation of Mankind, 1898; Three Discourses on Metaphysics, 1901; The Object of Life, 1904, and On Anthropomorphinism, 1904.

They are all pleasant reading—nothing particularly profound or startling, but withal very enjoyable. JELLIFFE.

JAHRESBERICHT ÜBER DIE LEISTUNGEN UND FORTSCHRITTE AUF DEM GEBIETE DER NEUROLOGIE UND PSYCHIATRIE, VIII. JAHRGANG. BERICHT ÜBER DAS JAHR. 1904. Redigiert von Prof. E. Mendel und Privatdozent Dr. L. Jacobsohn. Verlag von S. Karger, Berlin.

Twelve hundred pages of critical summary and abstract are to be in this eighth volume of this Jahresbericht.

Most of our readers are acquainted with the many excellent features of Mendel and Jacobsohn's yearbook, and in no essential feature has the present volume departed from those that have preceded it. It constitutes, as is well known, by far the best retrospect of the cognate branches published, and no neurologist or physicist who pretends in any way to keep posted in the modern literature of their specialty can afford to be without it. For the general consultant such a work is also indispensable..

We congratulate the editors and publishers alike for the continued excellence of this "yearbook."

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When the temperature keeps a point or two above normal for a few hours, the membrane of the oral cavity becomes dry and congested, causing great discomfort to the patient. Supplemented with this frequently comes the formation of sordes on the teeth and more or less inflammation along the marginal surface of the gums. The flow of saliva is checked and the sense of taste interfered with. In cases of this kind we may win the gratitude of the patient for all time by urging the frequent use of an alkaline mouth wash of the nature of Glyco-Thymoline. This solution is admirably adapted both by physiological action and therapeutic effect to meet the requirements. The normal flow of saliva is re-established, the further formation of sordes is prevented and the mouth is kept sweet and clean. No one can estimate the amount of comfort derived by the patient under this simple treatment.

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N. B.—Cut these out, paste on cards, and keep an index of American Neurological Work.

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" By weight, per litre	142.9
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" reduced after inversion, per litre	68 80
Sulphate of potassium, in grams, per litre	0.91
Tartrate, in grams, per litre	1.85
Ash	2.24
Acid, calculated as sulphuric	3.13
Acid sulphurous	none
Acid salicylic	none
Saccharin	none
Coloring matter	nothing abnormal
Deviation of polarimeter, before inversion	+2°44′
" " after inversion	−2°16′
Coca bases, combined, in grams, per litre	0.220

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   "Pediatrics," issue of November 1, 1900, pp. 349, 347, 338, 344.

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